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Methylmercury in Canada

volume 3

Exposure of First Nations and Inuit Residents to Methylmercury in the Canadian Environment

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METHYLMERCURY IN CANADA

Exposure of First Nations and Inuit Residents to Methylmercury in the Canadian Environment

A review of the Medical Services Branch, Health Canada
Mercury Program Findings to December 31, 1996

VOLUME 3

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INTRODUCTION

Since the early 1970s, the Medical Services Branch (MSB) of Health Canada has been involved in a methylmercury sampling program in First Nations and Inuit communities across Canada. Despite a considerable decrease in mercury levels among First Nations and Inuit populations, MSB continues to research the human health effects of mercury exposure. The rationale for MSB's continued involvement stems from the increased awareness of the health effects arising from low levels of mercury exposure.

Risk management experts and policymakers are reacting to the recently acquired knowledge concerning low levels of mercury exposure by revising mercury guidelines and consumption advisories established in the 1970s. The notion that separate guidelines are required for people who may be affected by lower levels of mercury, such as children and women of child-bearing age, is gaining acceptance.

Methylmercury in Canada, Volume III constitutes a closure to the formal methylmercury sampling program, but not the completion of MSB's activities in mercury research. The methylmercury program has analysed the results of 25 years of data from MSB's laboratory. Volume III summarizes the methylmercury program's most significant contributions to the knowledge of mercury exposure in Canada and related health issues affecting First Nations and Inuit populations.

The recently completed Effects on Aboriginals from the Great Lakes Environment (EAGLE) Project illustrates that mercury levels remain an important concern for First Nations communities. As mercury is one of the primary risk contaminants in Canada, MSB will continue to work with First Nations and Inuit communities to advance the frontiers of knowledge concerning mercury contamination.

Typical of the continued partnership between MSB and the Aboriginals Peoples of Canada are fish consumption guidelines which constitute an important factor in the restoration of traditional lifestyles in First Nations and Inuit communities. The guidelines advise which fish are unsafe for consumption and permit the traditional food consumer to make healthy diet selections by choosing fish with acceptable levels of mercury.

It is hoped that knowledge of mercury levels in populations throughout Canada will permit First Nations and Inuit Peoples to resume, or continue, eating the healthy foods that form an integral component of their cultural heritage.

STRUCTURE OF THE DOCUMENT

In the first two volumes, a particular order had been used following the general structure of scientific documents. As the objective of this third volume is to report to First Nations and Inuit communities on the results of the 25-year program, the document has purposely been structured to provide a general overview of the findings, followed by an overview of the state of scientific research in the field of mercury and human health. Finally, all the tables reporting results of the sampling program

are attached, providing the additional results since 1983 when volume 2 was released.

Although this document was prepared to be read and understood by the general population, we thought that it was also important to bring a scientific aspect to the document. Therefore, parts of the document are very technical. Nevertheless, most of the document is written in an easily understood manner.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS iii

INTRODUCTION v

LIST OF FIGURES ix

PREFACE xi

CANADIAN METHYLMERCURY RESULTS 1
- BACKGROUND 1
- TEST RESULTS TO DECEMBER 31, 1996 5
- TRENDS AND PATTERNS 9
- CLINICAL EXAMINATIONS 12
- SOCIO-CULTURAL (INDIRECT) EFFECTS OF METHYLMERCURY 14
- CANADIAN AND INTERNATIONAL METHYLMERCURY DEVELOPMENTS SINCE THE PREVIOUS REPORTS 19
- SUMMARY 21

HUMAN HEALTH RISKS FROM MERCURY 26
- THE MERCURY CYCLE 26
  - A. Introduction 26
  - B. Sources 27
  - C. Transport 28
  - D. Deposition 29
  - E. Levels in Media 29
  - F. Biotransformation and Bioaccumulation in Fish 30
  - G. Human exposures to Mercury 31

HUMAN HEALTH RISKS FROM METHYLMERCURY IN THE DIET 34
- DISPOSITION OF METHYLMERCURY IN THE BODY 34
  - A. Tissue Distribution 34
  - B. Metabolism to Inorganic Mercury 35
  - C. Pathways of Excretion 35
  - D. Biological Half-times 36
  - E. Mechanisms of Transport in Tissues 37
  - F. Blood Levels After Long-term Intake 37
- ADVERSE HEALTH EFFECTS OF METHYLMERCURY 39
  - A. The Clinical Pathology of Methylmercury 39
    - 1. Adult Exposure 39
      - a. Effects on the Nervous System 39

b. Other Effects 41
2. Prenatal Exposure 41
RISKS OF ADVERSE HEALTH EFFECTS 43
A. Risks from Adult Exposure 44
B. Risks from Prenatal Exposure 48
CONCLUSIONS AND RECOMMENDATIONS 56

HUMAN HEALTH RISKS FROM INORGANIC MERCURY IN THE DIET 58
Introduction 58
Human Exposure to Inorganic Mercury 58
Disposition in the Body 59
Adverse Health Effects 60
Risks of Adverse Health Effects 61

APPROACHES AND CONCLUSIONS BY REGULATORY AGENCIES AND ORGANIZATIONS 62
The Swedish Expert Group 62
World Health Organization 63
Food Directorate, Health Protection Branch (HPB) and the US Food and Drug Administration 63
US Environmental Protection Agency 64
Agency for Toxic Substances and Disease Registry (ATSDR) 64

REFERENCES 66

APPENDIX I 77

LIST OF FIGURES

Figure 1. Methylmercury levels: Human standards (MSB) 3
Figure 2. Trends in First Nations mean methylmercury levels (1974-1992) 6
Figure 3. Distribution of MeHg levels by northern cultural groups (1972-1987) 9
Figure 4. Seasonal variation of mercury level in a fishing guide from Grassy Narrows 10
Figure 5. Annual pattern of exposure to methylmercury in Canadian First Nations communities 11
Figure 6. Results of clinical examinations of individuals with MeHg levels > 100 ppb 12
Figure 7. Indirect impacts of mercury on human health and well-being 17

PREFACE

When the first volume of *Methylmercury in Canada* was written, almost 20 years ago, the Medical Services Branch methylmercury program was concentrating on clinical effects of methylmercury and the estimation of risk to individuals, using biological indicators, especially hair mercury levels (Wheatley et al., 1979). The assumption was that the higher the peak methylmercury level, the greater the risk to health. In conformity with that belief, hair samples were cut into 1 cm sample lengths to provide a temporal view. Reports on hair samples then presented the highest level found in any 1 cm of the total hair sample, as surrogate for level of risk for the individual for the period of time covered by the hair sample.

In more recent years, the program has continued to monitor the situation at a less intensive level, concentrating on follow up testing in communities where higher levels had been found, adding new communities as the opportunity presented, or on request by the communities. However, it has also become evident that although no provable direct clinical effects have been found in Canadian Native peoples (Wheatley et al., 1979; Wheatley and Paradis, 1996), there have been important indirect health effects in many communities linked to the socio-cultural and lifestyle disruption that occurred as a result of the presence of methylmercury in traditional food sources, especially fish and sea mammals (Wheatley, M.A., 1996; 1997).

This report updates data on mercury levels in individuals and communities, up to December 31, 1996, and presents analyses of those data, much of which has been published in peer review journals over the past few years (Wheatley and Paradis, 1995; 1996; Wheatley et al., 1997). However, it also includes discussion of the obserevd socio-cultural impacts of methylmercury, which in many cases may have had a greater impact on health than the so-called "direct" effects of mercury (Wheatley, M.A., 1996; 1997).

CANADIAN METHYLMERCURY RESULTS

BACKGROUND

The background to the Health Canada, Medical Services Branch (MSB) methylmercury program, internationally the largest human population methylmercury-level study done to date, is described in detail in the two previous volumes of this report. The first of these reports *Methylmercury in Canada* (Health Canada, 1979) discusses the relevant underlying human clinical and pathological effects of methylmercury (MeHg) in an international context, and provides and discusses the results of the Canadian program up to December 31, 1978. The second report *Methylmercury in Canada volume 2* (Health Canada, 1984) updates the findings to December 31, 1982. Both reports provide similar conclusions which will be further discussed later in this report.

Briefly, the underlying issues in Canada are that many Canadian Indigenous People continue, at least in part, to follow a traditional lifestyle, eating fish from lakes and rivers (Wheatley et al., 1979; Berkes, 1990) and, in the North, sea mammals such as seals and whales (Wheatley and Wheatley, 1988). These essential parts of the traditional diet may potentially expose these Native people, more than the general Canadian population, to environmental contaminants such as methylmercury, derived from whatever source, which bio-accumulate up the food chain in aquatic eco-systems.

This potential for high exposure and significant risk to health was first noted in Canada in the early 1970s in two Northern Ontario First Nations communities, Grassy Narrows and Whitedog. Both communities are on the English-Wabigoon river system into which effluent was being dumped by a chlor-alkali plant located upstream in Dryden (Rudd et al., 1983). The effluent contained mercury which was then methylated in the aquatic eco-system and bio-accumulated to significant levels (up to 24 μg/g) in the fish eaten by the people living at Grassy Narrows and Whitedog (Bishop and Neary, 1976). As a result of eating these fish, many residents of the two communities developed high blood mercury levels, including one individual who attained a level of 660 ppb (μg/L) in blood (Wheatley et al., 1979). Residents in the two communities were exposed through subsistence fishing and also because a number of them had prepared and eaten fish lunches while acting as fishing guides for visiting sports fishermen.

At approximately the same time, in northwest Quebec, levels of mercury in excess of 4 ppm (μg/g) were being found in some fish. In this case, the source of the mercury was less obvious, although it was believed to be at least partially industrial. The Cree First Nation people in this area were still living a relatively traditional existence, dependent on consumption of fish and wildlife. Also at that time, the first major damming of rivers under the James Bay hydro-electric development project was under way. The significance of damming became evident later when it was recognized that flooding of vegetation releases mercury into the aquatic eco-system (Bodaly et al., 1984). From the point of view of health authorities in the 1970s, the awareness was only that significant blood mercury levels were being found among the Cree people (Barbeau et al., 1976). A decision was made by MSB to develop an extensive program for assessing mercury levels among Native peoples across Canada, beginning with Grassy Narrows and Whitedog in Ontario and the James Bay Cree communities in Quebec, and then extending across the country (Wheatley et al., 1979).

The program did not begin as a research project. It was developed as a program for assessing exposure of Native people to methylmercury and the levels of risk produced by this exposure (Wheatley et al., 1979). Initially, the program was concentrated on individuals most likely to be at risk because of their lifestyle, such as guides, heavy fish eaters and those living a traditional lifestyle, including Inuit exposed to methylmercury through consumption of sea mammals. Following preliminary data release, a growing concern in communities led to a much wider sampling strategy in which all Canadian Native communities were invited to participate (Wheatley and Paradis, 1995).

It was rapidly realized that blood samples gave only a very limited amount of information, like a snapshot. A change was made to use hair samples as the biological indicator, making the assumption that hair grows at approximately 1 cm per month (WHO, 1976), and developing the technique of cutting the hair samples into 1-cm long segments. These were then analysed using the cold vapour atomic absorption method (Farant et al., 1981). This temporal view of exposure, going back as many months as the hair sample was centimetres long, was found to be critical to the program (Wheatley and Wheatley, 1981). Hair analyses gave both an estimate of the timing of the highest exposure during that period and also the length of significant exposure. In all hair samples, the results reported were the highest level found in any 1 cm of the sample, based on the assumption that the level of risk is related to the peak level reached. All analyses were carried out by the same MSB central laboratory using the same methodology throughout the program. The laboratory is

currently running the International Hair Mercury Quality Assurance (QA/QC) Program. For the sake of reporting consistency, earlier reports, having used blood mercury levels as the risk criterion, hair levels were converted to blood equivalents using a factor of 300, derived from Canadian First Nations data (Phelps et al., 1980). It was also noted that there was confusion among the Native communities and in the national media over the variety of units being used to report mercury levels. Therefore, again for clarity and consistency, it was decided to report hair levels in parts per million (ppm) and blood levels in parts per billion (ppb). Blood levels below 20 ppb (or 6 ppm in hair) were classified as being in the acceptable range and levels greater than 100 ppb in blood (30 ppm in hair) as "at-risk" (Wheatley et al., 1979). Clinical neurological examinations were offered to all individuals considered to be in the "at-risk "group.

Figure 1. Methylmercury levels: Human standards (MSB)

▸	Blood	< 20	ppb	Normal acceptable range
		20 - 100	ppb	Increasing risk
		>100	ppb	At risk
▸	Hair	< 6	ppm	Normal acceptable range
		6 - 30	ppm	Increasing risk
		>30	ppm	At risk

It is useful to place these levels in an approximate "order of magnitude" context. Thus, under 20 ppb is the acceptable range for methylmercury in blood, 200 ppb is the level at which the most sensitive 5% of adults may begin to show early signs of methylmercury toxicity such as paraesthesia (WHO, 1976), and at levels over 2000 ppb individuals show signs of serious methylmercury poisoning ("Minamata Disease").

The program was carried out through the MSB national network of nursing stations and health centres for Canadian Native people. The program activity peaked in the late 1970s and early 1980s and has since continued at a less intensive level, concentrating mostly on communities with significant exposure. Since the mid-1980s, the program among the Cree First Nation in Quebec has

been continued by the Cree Board of Health and Social Services (Chevalier et al., 1997) and, in the Northwest Territories (NWT), by the Government of the Northwest Territories (GNWT) Department of Health and Social Services following transfer of control of health services from the federal government to the GNWT in 1987.

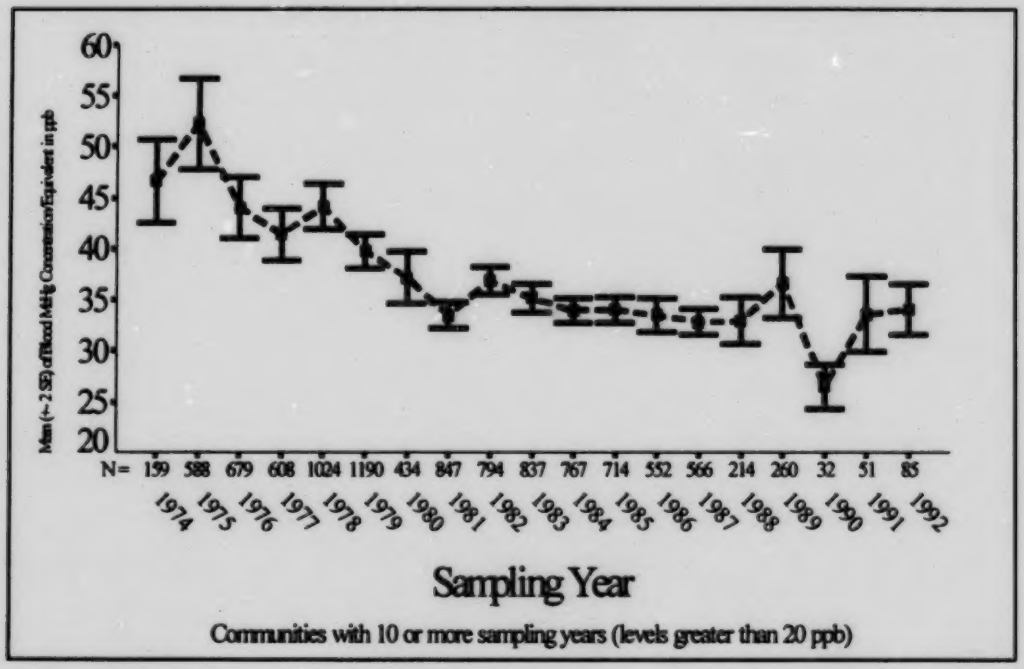
TEST RESULTS TO DECEMBER 31, 1996

Detailed results are presented in tabular form in Appendix I in the same format as in previous volumes. The cumulative testing data are set out in Table 1 in summary form, by region, for the period from January 1, 1983 to December 31, 1996 (i.e. continuing from the data contained in Volume 2) and in Table 1A for the whole period of the program from January 1, 1971 to December 31, 1996. Some individuals, especially those in the "at-risk" group, were re-tested over time. Therefore, an additional Table, 1B, provides a cumulative summary of numbers of individuals tested, grouped by highest level detected in that individual at any time and the region of that individual's residence. Table 2 sets out the cumulative testing results, by region and community, for the whole program period (1971-1996). The detailed tables (Table 3) cover the period 1983-1996 by year, region and community. Equivalent tables for the earlier years are contained in the first and second volumes of *Methylmercury in Canada*. The results for individuals with levels over 100 ppb (in blood or blood equivalent) are summarized for the 1983-1996 period, by region and level (Table 4) and for the whole period of the program in Table 4A. Table 5 provides individual results over 100 ppb, by community and level for the 1983-1996 period. Individual results by level over 100 ppb for the whole 1971-1996 period are contained in Table 5A. Table 6 sets out the total individual results over 100 ppb by region and year from 1971-1996. Table 7 provides a breakdown of individual levels over 100 ppb, by region and level for each year between 1983 and 1989, the last year in which a level over 100 ppb was detected in this program. The breakdown of results for individuals over 100 ppb, by sex, is given for 1983-1996 in Table 8 and in Table 8A for the cumulative 1971-1996 totals. Table 9 provides a breakdown of individual results over 100 ppb, by age group, and by region and community for the period 1983-1996. Table 9A does the same for the whole 1971-1996 period. Mother and child cord blood sampling results are summarized by region and level for the period 1983-1996 in Table 10 and for the period 1971-1996 in Table 10A.

In the period from January 1, 1983 to December 31, 1996, 13,876 blood or hair tests for methylmercury levels were carried out in 136 communities (Table 1). These tests brought the program totals to 72,556 tests in 529 First Nations and Inuit communities (Table 1A). This number represents testing of 40,634 individuals in the 529 communities (Table 1B).

The percentage of results at or above the "at-risk"level of 100 ppb has been steadily falling. This is immediately evident when comparing cumulative totals among the three time periods covered by the three volumes of *Methylmercury in Canada*. In the period up to December 31, 1978, 2.54% of results were in the "at-risk" range; between January 1, 1979 and December 31, 1982, that percentage dropped to 0.4% and in the most recent period from January 1, 1983 to December 31, 1996, dropped further to 0.3%. Between 1983 and 1996, 28 individuals had levels of 100 ppb or greater (Table 4) compared to 599 between 1971 and 1982. No levels greater than 100 ppb have been detected since 1989. There have been only two individuals in the MSB program with a level greater than 200 ppb detected in the 14-year period 1983-1996 (compared with 75 in the preceding 12 years). One was a resident of Resolute Bay, NWT in 1984 and the other was a resident of Cross Lake, Manitoba in 1989. Only occasionally have levels over 200 ppb been found later than 1989 in the Cree Board of Health program (Chevalier et al., 1997). These findings show the same trend of declining blood MeHg levels (Figure 2) reported by Wheatley and Paradis (1995).

Figure 2. Trends in First Nations mean methylmercury levels (1974-1992)



This graph, which includes mean mercury levels of individuals with over 20 ppb of mercury in blood or hair equivalent and communities with at least 10 sampling years, shows a general downward trend from 1974 to 1992. This trend was also confirmed in Grassy Narrows and Whitedog (Wheatley et al., 1997) and among the James Bay Cree in Quebec (Chevalier et al., 1997). It has yet to be determined whether the decrease in human mercury levels is due to actual decreases in the mercury levels of fish or because less fish is being consumed. It is possible that both of these determinants of human mercury levels are decreasing.

Over the duration of the program (1971-1996), by far the highest number of individuals with levels over 100 ppb (422) was found in Quebec, including both First Nations and Inuit. Next was Ontario (131) followed by the Northwest Territories (34) and Manitoba (19) (Table 4). However, as noted earlier, within the program no levels over 100 ppb have been found since 1989. In Saskatchewan, Alberta and British Columbia, no levels over 100 ppb have been found since the late 1970s, and in the Yukon and Atlantic Provinces none has ever been found (Table 6).

Although the highest peak levels found have been among First Nations peoples in Quebec and Ontario, the highest percentage of moderately elevated levels (between 20 and 99 ppb) were found in the Northwest Territories (Table 1A). Further analysis has shown this to be primarily among Inuit (Wheatley, B., 1994c).

Over the 25 years of the program, approximately 44% of those with levels over 100 ppb have been female (Table 8A). In the 1983-1996 period, this proportion fell to 10.7% (Table 8). In the critical child-bearing age group (15-45 years), the percentage levels over 100 ppb has reduced from 19.2% of individuals tested overall to 7.1% in the 1983-1996 period. This is a welcome finding, given the concern for subtle developmental effects on the fetus which will be discussed later.

Concern for fetal exposure led to a focus on maternal and fetal cord blood methylmercury levels in areas where elevated adult levels had been found. Comparison of Table 10 results (1983-1996) with Table 10A (cumulative program) shows that although the percentage of mothers with levels above the acceptable range has hardly changed (just over 6% of those tested), the percentage of cord blood (i.e. fetal) levels has been modified almost tenfold from 21.5% with levels above acceptable range in the overall program to 2.6% in the 1983-1996 period. Also, the peak levels found have reduced considerably, especially among children. In many cases, paired samples (mother/cord blood) were not taken, therefore, direct correlation between these two groups is not possible.

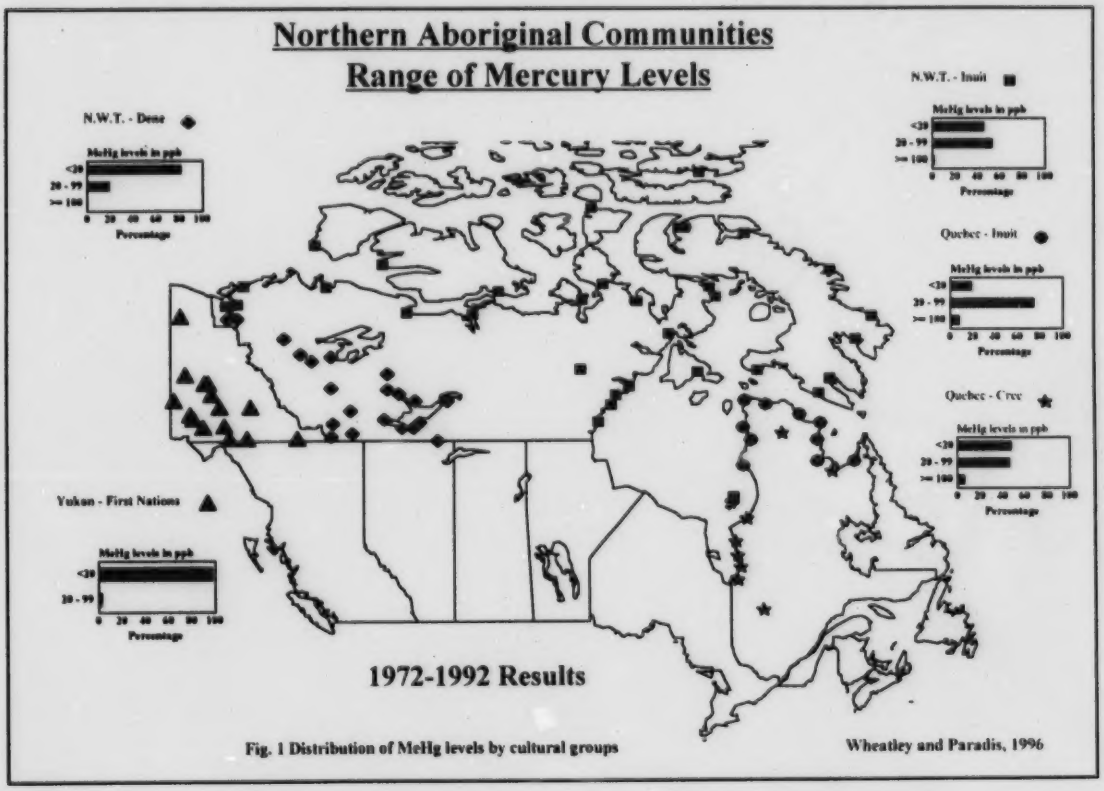
However, once again, the lower findings in recent years indicate a positive move and are consistent with findings from the Cree Board of Health and Social Services program (Girard and Dumont, 1995).

These are not sophisticated comparisons. Indeed, the data do not allow for complex analyses, as they have been collected over many years and in many communities by a diverse group of health professionals concerned with risk levels among their clients rather than for a hypothesis-based scientific study. Nevertheless, the weight of evidence from these tables of results is that methylmercury levels among the Native peoples of Canada are now considerably lower than when the program started over 20 years ago.

TRENDS AND PATTERNS

The general downward trend in methylmercury levels among Canadian Native peoples was discussed in the previous section. However, more detailed analyses, separating out groups with distinct, different lifestyles, has shown some variations on this general pattern. For example, the graph shown in Figure 2 represents mercury levels among First Nations peoples south of 60^0 N. When the mean community levels were graphed for the N.W.T. Inuit alone, there was no trend of any note for the period from 1975-1987 (Wheatley and Paradis, 1996). Similarly, comparison of mean community level patterns among the N.W.T. Inuit and Dene, Yukon First Nations and Quebec Cree and Inuit showed a much higher percentage in the 20-99 ppb range among Inuit than among Dene and other First Nations groups, as shown in Figure 3 (Wheatley and Paradis, 1997).

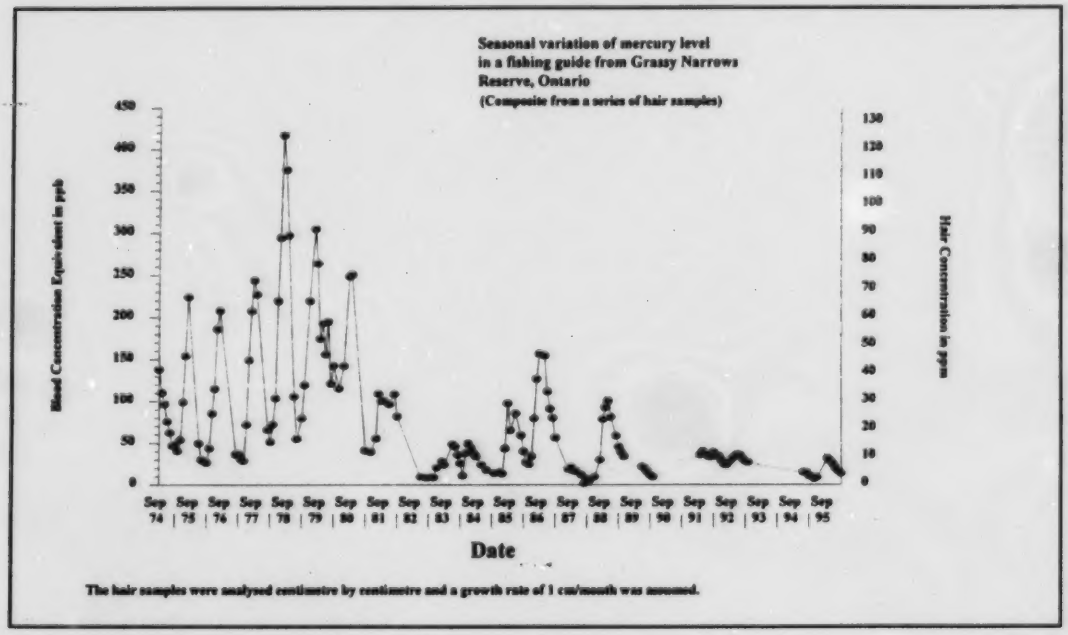
Figure 3. Distribution of MeHg levels by northern cultural groups (1972-1987)



When the mean community level results for the years up to 1992 were analysed by dividing them into quartiles for comparison purposes, it was found that the fourth quartile (i.e. highest mean levels) contained primarily northern First Nations and Inuit communities (Wheatley and Páradis, 1995). When the quartile approach was repeated for the N.W.T. data (1972-1987) alone, the quartile values, overall, changed upward, indicating a general higher exposure. The fourth quartile (range 30-48 ppb) included primarily the northern and eastern Inuit communities, many of which continue to follow traditional lifestyles with their residents consuming fish and sea mammals (Wheatley and Paradis, 1996).

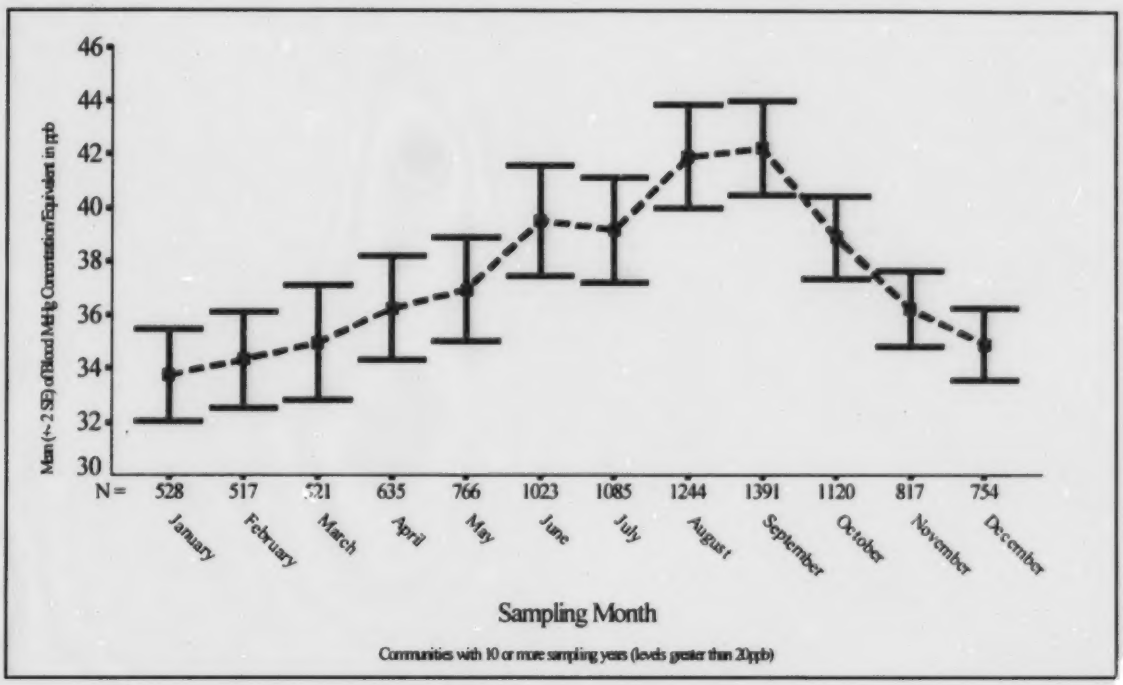
Seasonal exposure of Canadian Native people has caused concern over the years. At the individual level, there is a record of over 20 years of seasonal exposure in a guide from Grassy Narrows who, in earlier years, reached high mercury levels each summer – the highest being 430 ppb in 1978, but returned to acceptable levels each winter (Wheatley et al., 1997) (Fig. 4).

Figure 4. Seasonal variation of mercury level in a fishing guide from Grassy Narrows



At the community level, the mean peak blood mercury levels in individuals with levels over 20 ppb (i.e. presumed fish eaters) in First Nations communities (not Inuit) with 10 or more sampling years showed an obvious seasonal pattern, with a peak in early fall (Wheatley and Paradis, 1995) (Fig. 5).

Figure 5. Annual pattern of exposure to methylmercury in Canadian First Nations communities.



Again, the same process was followed with the N.W.T. Inuit data alone because of the unique Inuit lifestyle. The result was a double peak in the spring and fall which fitted with Inuit fishing and hunting patterns (Wheatley, B., 1994c; Wheatley and Paradis, 1996). However, it is still unknown whether effects, if any, from these seasonal peaks are cumulative or whether returning on a regular basis to acceptable levels counters any negative health effects due to the peak levels.

CLINICAL EXAMINATIONS

As mentioned earlier, in the initial program set up in the mid-1970s, all individuals with levels greater than 100 ppb were offered full neurological examinations. Following recommendations in the first volume of *Methylmercury in Canada* (Wheatley et al.,1979), this was modified to offering examinations to those with levels over 200 ppb. Not all accepted neurological examination, but, as shown in Figure 6, of the 99 individuals who did accept and were clinically examined by very experienced neurologists, 61 had no significant abnormalities, 27 were found to have some abnormalities not attributable to methylmercury, and 11 had neurological findings possibly attributable to methylmercury. No definitive diagnosis of methylmercury poisoning could be made (Wheatley and Paradis, 1996).

Figure 6. Results of clinical examinations of individuals with MeHg levels > 100 ppb

Number examined	99
No significant abnormality	61
Abnormality not attributable to MeHg	27
Findings possibly attributable to MeHg	11
Definitive diagnosis	0

This diagnostic difficulty was a challenge throughout the program even when levels of methylmercury were high compared to present levels (Wheatley et al., 1979).

In the umbilical cord blood sampling program, the highest cord blood level found was 224 ppb and 523 samples (21.8%) were found to have levels greater than 20 ppb. However, neurological examinations performed on the children showed no findings that could be attributed to methylmercury (Wheatley and Paradis, 1996). A follow-up study of this group of children has been initiated to try to assess more subtle developmental effects and educational attainment (Wheatley et al., 1997).

An earlier study, funded by Health Canada in 1979, was carried out by McGill University, among the Crees of James Bay in northern Quebec. In that study, although an association was noted between findings on clinical examination of tone and reflexes in Cree boys and the concentration of methylmercury in their mothers' hair during pregnancy, the abnormalities were very mild and in most children it was difficult to be certain that they represented abnormal function (McKeown-Eyssen and Ruedy, 1983).

As noted elsewhere, and in the first volume of *Methylmercury in Canada* (Wheatley et al., 1979), the relationship between peak mercury levels and neurological findings possibly attributable to mercury was random. Whether these neurological findings related to differences in individual sensitivity or to earlier non- recorded methylmercury levels cannot be ascertained. No dose-response relationship could therefore be established from these data. The mercury levels (over 200 ppb) which were found in a small percentage of individuals were sufficient to identify a significant health risk and to lead to the expectation that the most sensitive individuals may show early or mild symptoms of methylmercury poisoning (WHO, 1976). It could also be anticipated, on the same theoretical basis, that at the levels found in Canadian Native peoples (Table 1A), the differential diagnosis from other conditions would be difficult. This was borne out in practice by the clinical findings. While it can be stated categorically that severe methylmercury poisoning ("Minamata Disease") has not been found in Canada, milder forms of mercury poisoning, although difficult to prove conclusively (Wheatley et al., 1979), possibly have occurred (Fig. 6). However, with the lower levels currently being found, even mild forms are unlikely to occur in adults at the present time.

SOCIO-CULTURAL (INDIRECT) EFFECTS OF METHYLMERCURY

The preceding sections have discussed the direct effects that occur, or may occur, when high levels of mercury pose a danger to health. However, as outlined, despite the extensive testing and examinations throughout the life of the program, no direct effects on human health have been proven among Canadian Native people (Wheatley et al., 1979; Wheatley and Paradis, 1996). On the other hand, health and well-being have been seriously affected by what, from an epidemiological point of view, are considered to be indirect effects. These are the social and cultural impacts on Native peoples which may result when contaminant levels are high, or are perceived to be high.

As stated earlier, many Canadian First Nations and Inuit people still follow traditional lifestyles. These lifestyles revolve around the harvesting of natural resources to supply basic subsistence needs, especially food, but harvesting activities also provide a forum where traditional values and life skills can be passed from one generation to the next. Harvesting is therefore not a separate activity but an integral part of the cultural existence of Native people (Berger, 1977). Canadian Aboriginal people perceive themselves to have a special relationship with the environment (McClellan, 1987) and this special relationship encompasses the responsibility to look after the environment for the benefit of future generations (Wheatley, M.A., 1996; 1997). Maintaining a healthy environment is therefore seen as a necessary prerequisite to ensure good health for both individuals and communities (Wheatley, M.A., 1994).

The activities associated with the harvesting of country or traditional foods have both nutritional and non-nutritional benefits (Dumont and Kosatsky, 1991; Berger, 1997). On the nutritional benefit side, extensive research in Canada has shown that traditional foods provide high protein, low carbohydrate diets rich in essential minerals and nutritionally superior to store-bought foods (Kuhnlein, 1995; Kuhnlein et al., 1996; Receveur et al., 1997). The non-nutritional benefits range over the social, economic, cultural and spiritual aspects of everyday life (Wheatley, M.A., 1996). As well as providing good exercise, many harvesting activities bring people together and promote social cohesion (Berger, 1977). They also provide a learning experience for young people, teaching them skills for use in later life, with good hunters and fishermen providing role models (Wheatley, M.A., 1994; 1996). On the economic side, these skills go beyond subsistence needs and

provide a basis for employment opportunities such as guiding and outfitting. Harvesting activities offer an opportunity to share cultural values and beliefs and transmit them to younger generations, providing a demonstration of the culturally important values, which enable people to reinforce their special relationship with the environment (Wheatley, M.A., 1994; 1995; 1996;1997). Disruption of this world view and pattern of living can therefore cause a major disruption to the health and well-being of both individuals and communities (Shkilnyk, 1985; Johansen, 1993; Erikson, 1994;).

In urban societies, store-bought food safety is regulated by inspection at the production or processing stage. Consumers rely on this for protection. When country foods are contaminated to a level that is potentially hazardous to consumers, Native or non-Native, the only protective measure that can be offered is to provide advice to limit consumption (Wheatley et.al.,1979). Providing advice against consumption to Native peoples, however, is tantamount to advising against fishing and hunting. A complex reaction follows. Subsistence activities are an integral part of Aboriginal peoples' cultural identity, forming a social system and not merely providing a staple diet. The identification of contaminants in country foods therefore understandably causes consternation in Native communities, and, in the absence of clear and understandable information, fear and suspicion may lead communities to make decisions based on inaccurate or incomplete information.

Although a variety of factors contributes to social disruption in Canadian Native communities at the present time, the knowledge that contaminants such as mercury are affecting traditional food sources compounds the situation. In some situations, this is a major factor, as was the case in Grassy Narrows and Whitedog in Ontario and Sugluk, now Salluit, in northern Quebec. In Grassy Narrows and Whitedog, there was no alternative but to advise non-consumption of locally caught fish (Wheatley, 1979). However, the advice had long-term repercussions. These were the first communities in Canada to be identified as affected by methylmercury and investigated. Answers were often slow in coming and comparisons were therefore readily made to the outbreaks of Minamata Disease in Japan, despite an order of magnitude difference in exposure in most cases. The community perception was that information was being withheld by scientists or government officials or was presented in a language not easily understood by communities (Shkilnyk, 1985). Even with a replacement supply of fish in the communities, there was a change in diet because the fish supplied was a different species from the preferred locally caught pickerel. There was also a change in

lifestyle, which had previously centred around the harvesting of fish. The substituted fish taken from a freezer could not replace the social activity of harvesting for family needs, nor compensate for the negative economic impact of the closing of the commercial fishery. The indirect effect of mercury was therefore social and cultural disruption and severe socio-economic damage (Usher et al., 1979; Shkilnyk, 1985; Erikson, 1994). Erikson (1994) reported that between 1974 and 1978, Grassy Narrows, with a population of 400, recorded 31 violent deaths, including 5 suicides, most of which were alcohol related. The consequences still appear to be affecting these communities (Guttormson, 1995). In another situation, the Crees of James Bay found the information provided by health workers or the media to be alarming or contradictory. Some chose to ignore the information whereas others drastically changed their lifestyles (Dumont and Kosatsky, 1991).

In Sugluk (Salluit), the response was more immediate. Sensational, inaccurate media reporting which exaggerated the possible danger from methylmercury led residents in this Inuit community to stop eating the country foods on which they depended (Wheatley and Wheatley, 1981). The extent of this dependence was reflected in harvesting data (James Bay and Northern Quebec Native Harvesting Research Committee, 1979). In remote areas, there are few available, affordable, acceptable, alternative foods. In the case of Salluit, media references to "Minamata Disease" created a crisis situation, before an investigation into the reason for the relatively high human mercury levels which had been identified could be carried out. The greatest problem in Salluit was the fear produced by a lack of understanding of risk levels of methylmercury (Wheatley and Wheatley, 1981).

Aboriginal peoples' assessment of a situation is thus determined by perceptions that are based on traditional values, beliefs and observations. In the general population, research has shown that risks which are perceived as imposed on individuals, risks perceived to be unfairly shared or not easily controlled by individuals, and risks associated with human-made hazards all appear greater than more familiar risks and those that are undertaken voluntarily (Slovic et al., 1982). Many environmental contaminants, including mercury, being invisible and difficult to understand, have all these characteristics. This therefore makes them especially threatening for Aboriginal people whose concepts of health and environment are interrelated. The perceived threat may then lead to actions that have a major impact on health and well-being (Wheatley, 1996). Fears and concerns about the safety of traditional foods may result, as stated earlier, in a change of diet and lifestyle,

socio-economic damage, and social and cultural disruption as summarized in Figure 7 (Wheatley and Wheatley, 1997).

Figure 7. Indirect impacts of mercury on human health and well-being

CHANGE IN DIET
CHANGE IN LIFESTYLE
SOCIO-ECONOMIC DAMAGE
SOCIAL/CULTURAL DISRUPTION

▼

OBESITY, DIABETES
LOSS OF FITNESS
FAMILY BREAKUP/COMMUNITY VIOLENCE
ALCOHOLISM, DRUG ABUSE, SUICIDE

The change in diet, from high protein fish or sea mammals to high carbohydrate foods and the associated change from an active, socially cohesive lifestyle to a sedentary, less social lifestyle contribute to obesity, loss of fitness (Rode and Shephard, 1994) and an increased incidence of adult onset diabetes (Young et al., 1990), which in some areas, has risen from zero 50 years ago to four times the Canadian average today (Wheatley, B., 1994a). Socio-economic damage occurs with the loss of both the subsistence economy and the wider economic base, especially in communities where fishing was a central component of the lifestyle, as it was in Grassy Narrows and Whitedog and in the James Bay Cree communities. This socio-economic damage may then lead to social and cultural disruption, because the opportunity to be role models as good providers or to share and pass on these and other cultural values has disappeared. The breakdown in traditional roles and relationships associated with traditional lifestyles and the consequent loss of control over many aspects of life has been associated with violence, gambling and smuggling on the Mohawk reserve of Akwesasne on the St. Lawrence River (Johansen, 1993). This situation has also contributed to the increased community and family violence, and increased alcoholism, drug and substance abuse and suicide witnessed in the Grassy Narrows and Whitedog reserves (Erikson, 1994; Guttormson, 1995).

Residents of Grassy Narrows and Whitedog saw the environment as poisoned and could no longer rely on their traditional knowledge and skills to guide them. The persistent social problems in these communities reflect the hopelessness that results from the destruction of a familiar lifestyle and the loss of self-esteem and traditional values. What began as a potential health problem, because of high mercury levels in local fish, became a much greater social and cultural problem. In Salluit, the incident described shows how poor, inaccurate and misleading media information can exacerbate a problem which is already difficult to understand and is perceived as a threat. In other communities, like Akwesasne, the breakdown in traditional roles and relationships has contributed to increased violence in the community. It is therefore vital, when making policy decisions such as risk levels and guidelines, to be aware of and take into account the possible socio-cultural impacts of these actions and decisions.

CANADIAN AND INTERNATIONAL METHYLMERCURY DEVELOPMENTS SINCE THE PREVIOUS REPORTS

Mercury as a global pollutant, and its effects on human health, continues to have a high profile internationally and it has been the subject of biennial international conferences since 1990 (Porcella, Huckabee and Wheatley, 1995; Wheatley and Wyzga, 1997). It has also been the subject of ongoing discussion by the World Health Organization (WHO, 1990; 1994; 1997).

The overall international viewpoint, together with a technical discussion, is presented in a separate section. From the Canadian point of view, the critical international development in recent years has been the increase in concern for subtle developmental effects on the fetus in utero at maternal methylmercury levels in a range that has been seen in a significant percentage of Native women of child-bearing age in Canada (Wheatley and Paradis, 1996).

The fetus is more sensitive to methylmercury than the adult. The WHO Expert Committee on methylmercury noted, in 1990, that a 5% risk of subtle developmental effects may be associated with a peak mercury level of 10 to 20 μg/g (ppm) in maternal hair (WHO, 1990). Overall, the MSB study of methylmercury levels in Canadian Native people found 1495 women in the 15-45 year age group (13.7% of the women in this group who were tested) with levels greater than 10 μg/g in hair. The NWT component of the study showed that between 1972 and 1987, over 30% of the Inuit women in this age group who were tested had levels over 10 μg/g (Wheatley and Paradis, 1996). Therefore, based on the 1990 WHO criteria, a risk exists, or may have existed, for the fetuses of a number of Native women in Canada, dependent on coincidence of time, exposure and pregnancy.

In the context of international health, the question can be raised why the potential for small neuropsychological effects of methylmercury on the fetus may be important. From the strictly medical viewpoint, the response will be that if there is risk of any neurological damage to adults or children, then suitable action, such as advice on reduction of intake, must occur. From the broad public health or population health point of view, as distinct from concerns for the individual, the reaction will be that even small effects on fetal development, at a population level, may have serious long-term impacts on the achievement and quality of life of that population group as a whole.

On the other hand, many of the potentially affected peoples live in remote areas, such as northern Canada and Amazonia, and receive exposure to methylmercury from food sources, especially from fish, upon which they depend as their primary source of nutrition. Fish is also an essential part of the diet of many island populations throughout the world. Many of these populations are already undergoing a lifestyle transition, with increases in cardiovascular and other diseases such as diabetes, because of changing diets. Any reduction in fish consumption may have a potentially serious impact on morbidity and mortality.

In addition to the direct medical effects referred to above, Canada has been the first to raise, in international fora, concerns over the indirect health effects of methylmercury on Native peoples due to socio-cultural and lifestyle disruption precipitated by mercury and other contaminants in traditional food sources (Wheatley, B., 1994a; Wheatley, M.A., 1995; 1996; 1997). In the Canadian experience, as discussed in an earlier section, these indirect effects have been very important, and on occasion, have produced a potentially more serious health problem than that posed by direct clinical effects. These issues are now receiving international attention (Wheatley and Wheatley, 1997).

SUMMARY

Both previous volumes of *Methylmercury in Canada* included a number of conclusions, many of those in the first volume being subsumed into volume 2. In many ways there have been few changes, other than the number of results and the number of communities in which testing has occurred. Among the most important general conclusions were the following (conclusion numbers taken from the first volume of *Methylmercury in Canada*):

Conclusion 11:

> *"The relationship between peak mercury levels and neurological findings possibly attributable to mercury was random. No dose-response relationship could be established from these data."*

Conclusion 12:

> *"The mercury levels being found in a small percentage of individuals are sufficient to identify a significant health risk and to lead to the expectation that the most sensitive may show early or mild symptoms of methylmercury poisoning. However, it can also be anticipated, on the same theoretical basis, that at this level the differential diagnosis from other conditions will be difficult. This is borne out in practice by the clinical findings."*

As discussed earlier, these conclusions continue to be valid.

The statement in Conclusion 13:

> *"While it can be stated categorically that severe methylmercury poisoning ("Minamata Disease") has not been found in Canada, milder forms of mercury poisoning, although difficult to prove conclusively, quite possibly are occurring."*

Conclusion 13 also remains true, although with the lower levels currently being found, even mild forms are unlikely in adults.

Conclusions 14 and 15 relate to fetal exposure which continues to be a concern and has been the subject of further work, as discussed earlier and by Wheatley and Paradis (1995; 1996). Some initial work has been carried out relating to the longitudinal study of prenatally exposed children as recommended in Conclusion 20 (Wheatley et al., 1997).

Conclusion 21 states:

> *"It is important that a careful balance be established between the advantages of the traditional Indian and Inuit way of life, which can provide an excellent source of dietary protein in the form of fish and sea mammals, and the potential danger to health due to elevated mercury levels in that food source. There must be an on-going commitment to work with communities and individuals to devise local solutions, tailored to specific circumstances, needs and lifestyles."*

This was the initial identification of this socio-cultural reality and has been the basis for a considerable volume of further work (Wheatley, B., 1994a; Kuhnlein et al., 1995; Wheatley, M.A., 1996; 1997). Further discussion is contained in earlier sections of this volume.

Conclusion 24 (i-iv) noted the need for further research in a number of areas, including:

> 24 (i): *"The possible relationship between acid rain, airborne mercury and environmental mercury in areas remote from direct industrial contamination."*

A great deal of research has been and continues to be performed in these areas and continues to be the basis for animated debate, especially concerning the relationship between anthropogenic and natural sources (Wheatley and Wheatley, 1988; Muir et al., 1992).

> 24 (ii): *"A more detailed assessment of the almost unique seasonal pattern of exposure to mercury seen in Canadian Indians, and whether health effects may be cumulative or reversible."*

Detailed assessments of these exposures have been carried out (Wheatley and Paradis, 1995; 1996). The issue of cumulative effects or reversibility still remain unresolved. However, it is reasonable to assume that the seasonal pattern of exposure, with human blood MeHg levels returning to near normal in winter, has prevented continuous cumulative exposure which could have led to severe methylmercury toxicity in some cases.

> 24 (iii): *"Possible synergistic or antagonistic interactions between mercury and other substances in the environment, including selenium."*

The issues of synergism and antagonism between mercury and other substances, including selenium, remain of considerable interest, but also remain unproven and a matter of conjecture in humans (Magos, 1991).

> 24 (iv): *"The identification of a sensitive specific biochemical test capable of acting as an 'early warning' indicator of metabolic effects of methylmercury, and thereby able to replace the present somewhat crude assessment, based on blood or hair mercury levels and clinical examinations, which does not allow for individual sensitivity."*

There is still no progress in this area although a bio-indicator of this type continues to be needed.

The maximum acceptable level of methylmercury in fish for commercial sale in Canada, based on assumed average consumption of fish, was set at 0.5 ppm before the MSB methylmercury program began in the 1970s and remains to the present day. In the 1970s, however, as noted in the first volume of *Methylmercury in Canada* (Wheatley et al., 1979), the concern for health effects on Native peoples, due to the quantities of fish being eaten by them, was so great that MSB felt obliged to recommend 0.2 ppm as the maximum acceptable level of methylmercury in fish being consumed by Native peoples. This level was based on the realities of the times, and, as a recommendation, can be changed at any time to reflect changes in fish consumption patterns.

Given the present state of knowledge and the levels of methylmercury being currently found in Native peoples in Canada, it would be reasonable to say that the direct risks to the health of adults due to methylmercury are minimal at this time. However, the indirect health risks due to socio-cultural and lifestyle disruption and changed eating patterns remain of concern. The examples cited earlier illustrate the need for good communication with affected individuals and communities, and the consideration of many factors when providing advice and developing policy. To minimize impact on communities, this means recognizing the importance of communication at all stages of the investigation (Wheatley, M.A., 1995; 1996) and involving the communities in defining solutions (Dumont and Kosatsky, 1991). When animal or human testing is undertaken, people should be told why tests are needed, how they will be done, what they can and cannot do, and they should be provided with an explanation of what the results mean in non-technical language. Understandably,

non-technical language which provides non-contradictory information is important to help reassure communities if there is no risk, to prevent panic where a possible risk exists, as was the case in Salluit, or to justify advisory notices if they are necessary, as was the case in Grassy Narrows and Whitedog. Non-technical language includes the use of understandable units of measurement, which have relevance for the people concerned, such as "number of fish meals per week." Good communication at all stages of the investigation is essential in order to minimize the socio-cultural effects discussed above which, in turn, reduces the adverse effect these impacts have on health. It is crucial to balance carefully all aspects of the situation, including social and cultural, when providing advice that may have an impact on the lifestyle and thus on the health and well-being of the Aboriginal peoples concerned.

Estimates of health risk from methylmercury in fish have not changed substantially since the publication of previous volumes of *Methylmercury in Canada*. New reports of studies in adults (Spitzer et al., 1988) and Lebel et al. (1995, 1998), raise the possibility that adverse effects may occur at hair levels below 50 ppm but no adverse effects have yet been demonstrated at or below 30 ppm, the current "at-risk" level adopted previously by MSB (Figure 1).

Risks to the fetus through exposure in utero are still of concern, although no adverse effects have been reported below the current normal acceptable range of less than 6 ppm in hair. Two new studies have been reported on populations having high consumption of ocean fish in Peru (Marsh et al., 1995) and in the Seychelles Islands (see Davidson et al.,1998 for the most recent report). Both studies covered a range of hair levels up to about 30 ppm in hair without finding an adverse effect of methylmercury on child development. However, reports of a study in the Faroe Islands where exposure to methylmercury is from consumption of pilot whales claims to have found adverse effects on child development at maternal mercury levels equivalent to hair levels of 10 ppm (Grandjean et al., 1997; 1998). However, more information is needed on this study before a definite conclusion can be made that effects are actually occurring at such low levels. All other studies to date of prenatal exposure to methylmercury in fish have not shown effects below 10 ppm.

In conclusion, the risks levels for methylmercury reported in Figure 1 still seem to be appropriate for Canada. Thus, the normal acceptable range is less than 6 ppm in hair, equivalent to 20 ppb in whole blood. The level of 30 ppm in hair is still the best approximation to the "at-risk"

level, with the caveat that this level may be changed depending on the results of ongoing studies in the Seychelles and the Faroe Islands. There is also an ongoing study being carried out in northern Quebec by Laval and Wayne State universities which is looking at prenatal mercury and PCB exposure and various developmental endpoints.

Inorganic mercury is also present in the Canadian diet. Most of this may originate from the breakdown of methylmercury in fish, as fish meal is used as fodder for livestock. The methylmercury is metabolized in animal tissues to appear as inorganic mercury in the diet. Livers in seals and other fish-eating marine mammals contain very high levels of inorganic mercury. It appears to be in an inert form, not posing any toxic hazards to the seal or to humans consuming the liver. In general, health risks appear to be minimal although infants, including suckling infants, may acquire higher body burdens than adults.

Public health risk management decisions and the provision of responsible public health advice require the use of a very difficult and complex risk–benefit equation. This must concentrate on the need to balance the theoretical basis of the risk assessment, for different population groups and for different exposure patterns, against the impact on health and well-being that may result from a risk management strategy which includes advice to restrict consumption of traditional foods, especially fish. The emphasis must be on the irreversibility of adverse neurological effects. Mild, transient effects are of some public health concern but they do not give sufficient reason to provide advice that will affect the diet, lifestyle and ultimately the health of Canadian Native peoples to whom the advice is given. At the present time, except at very high methylmercury levels, certainly higher than those currently being found among Native peoples in Canada, there are no straightforward answers.

HUMAN HEALTH RISKS FROM MERCURY

THE MERCURY CYCLE

A. Introduction

Mercury is aptly named after the messenger of the Roman gods. It is highly mobile physically, chemically and biologically. This high mobility is important to understand the movement of mercury from its environmental sources to humans. Mercury is the only metal that exists in the liquid form at room temperature. It readily volatilizes as a monatomic gas, Hg^0, usually referred to as mercury vapour. This gas is highly mobile and, as discussed below, is the principal form of mercury transported in the atmosphere. The metallic or elemental form of mercury can lose one or two electrons to form mercurous and mercuric respectively

$$Hg^0 \text{ ->> } Hg\text{-}Hg^{++} \text{ ->> } Hg^{++} \quad \text{..... (1)}$$

Both mercurous and mercuric ions form a variety of chemical compounds with mercuric mercury forming the most important compounds in terms of the environment. The mercuric ion can bind with carbon atoms to form organo mercury compounds. Environmentally, the most important are the monomethylmercury compounds, CH_3HgA, containing the methylmercury cation, CH_3Hg^+. We will refer to the methylmercury compounds, CH_3HgA, collectively as "methylmercury" except where the attached anion, A^-, is known and plays a special role.

A chemical reaction of special importance is the reaction of either Hg^{++} or CH_3Hg^+ with the sulfydryl anion either as $S^=$ or ^-S-R where the moiety, R, is usually a protein or amino acid.

Examples are: $Hg^{++} + S^= = HgS$.....(2)

where HgS, known as cinnabar, is the insoluble form usually found in mercury ores, and in anaerobic sediments in fresh and ocean water;

and the reaction $CH_3Hg^+. + {}^-S\text{-}R = CH3Hg\text{-}S\ R$.....(3)

which dominates the chemistry of methylmercury in biological tissues and fluids. The reaction is

rapidly reversible, thus allowing the methylmercury cation to "jump" from one sulfydryl group to another giving it a high chemical and biological mobility. A detailed discussion of mercury chemistry can be found in Nriagu (1979).

Human exposures from both natural and anthropogenic sources of mercury follow the general sequence: sources -> transport-deposition -> environmental levels -> methylation by microbiota and subsequent bioaccumulation in fish and fish-eating sea mammals -> human consumption (Fitzgerald and Clarkson, 1991). The ensuing text will follow this sequence.

B. Sources

The sources of mercury are usually distinguished as natural versus anthropogenic, on the assumption that the latter is subject to controls. These two sources are estimated to make roughly equal contributions to current emissions to the atmosphere (Lindqvist et al.,1991). The anthropogenic sources may be subdivided into those industries that specifically use mercury and its compounds, such as in the manufacture of dry batteries or in the chlor-alkali industry, versus those activities where mercury is inadvertently released, such as in coal-burning power stations and municipal waste incinerators. Anthropogenic sources release mercury to the atmosphere in the form of mercury vapour and possibly in a "reactive form" whose chemistry is not well understood. In fact, no speciation studies of the form of anthropogenic mercury released to the atmosphere have yet been reported for the American continent, the available data coming from Europe.

The use of mercury by industry has fallen dramatically in developed industrialized nations. For example, mercury uses in the United States have fallen by at least 50% since 1990. Release from human sources also occurs as run-offs and discharges directly into rivers, lakes and ocean bays. Appreciable local pollution has occurred by this route, for example, discharges from chlor-alkali plants and paper pulp plants in certain areas of Canada. Such discharges have essentially ceased but the residual mercury remains in the aquatic sediments. Eventually, the mercury-contaminated layer may be covered by layers of fresh sediment that effectively immobilizes the contamination.

Approximately 4000 tons of mercury are released worldwide each year, from anthropogenic sources, of which about one-half is released as the vapour to distribute globally. The other half is as yet poorly defined reactive forms that distribute locally and regionally. Compared to the U.S.

contribution of 200 to 300 tons annually, the Canadian contribution is about 20 tons (Toronto Conference, 1997).

The main contributors to anthropogenic releases are coal-burning power stations and municipal and medical waste incinerators, followed by a host of minor sources. The release of mercury from dental offices due to the widespread use of mercury amalgam tooth fillings has now been recognized as an important source in municipal sewers.

The so-called natural sources also have two important divisions: 1) the true natural emissions of mercury geologically present in the Earth's crust such as mercury-rich soils and rock as well as emissions during volcanic activity, and 2) re-emission of mercury previously deposited on the earth's surface from anthropogenic and perhaps natural sources. For example, it has been estimated that some 200,000 tons of mercury from anthropogenic sources have been deposited on the earth's surface in the past century (Expert Panel, 1994). The re-emission of only a small fraction of this historic load would easily match current anthropogenic emissions.

The "ping-pong" type of mobility of mercury – emission-deposition-re-emission – is a special aspect of the physical mobility of this metal connecting the historic emission to current mercury load in the atmosphere. Generally speaking, these "natural sources" both for geological and historic mercury are believed to be about equal to anthropogenic sources. The form of mercury from natural sources is entirely mercury vapour and, as such, will be distributed globally.

C. Transport

Although release of mercury into waterways from soil erosion and human activity is an important pathway, the atmosphere is the medium through which mercury is globally transported and distributed (Fitzgerald and Clarkson, 1991). Mercury vapour has a residence time in the atmosphere of about one year (Lindqvist and Rodhe, 1985). This allows sufficient time for mercury vapour emitted from any point source on the earth's surface to travel on a global scale.

The background level of total mercury is estimated at about 1.6 ng Hg per cubic metre of air. Over 90% is present as mercury vapour. Traces of monomethyl and dimethyl compounds, CH_3HgCH_3, have been detected in atmospheric samples. Mercury in the particulate form accounts

for less than 1% of atmospheric mercury. The total amount of mercury in the earth's atmosphere is estimated to be about 5000 tons.

D. Deposition

The 2000 tons of reactive mercury entering the atmosphere from anthropogenic sources does not enter the global cycle. It is deposited in rain water and as dry deposition as particulates. The other 2000 tons, emitted as mercury vapour, must first be converted to divalent mercury to form water-soluble compounds that are deposited in rain water. The chemical mechanism of the oxidation reaction is still unknown but it may occur, in part, on water surfaces where the so-called oxygen-water couple is known to oxidize Hg^{o} to Hg^{++}.

As discussed above, the deposition of mercury on land or water can result in a ping-pong-like sequence of re-emission, transport and re-deposition. The Hg^{++} deposited in natural water or soils can be reduced back to the volatile Hg^{o}, and thereby return to the atmosphere. The depths of the ocean are regarded as the ultimate sink as Hg^{++} falls to anaerobic regions where it reacts with reduced sulfur (the sulfide anion, $S^{=}$) to precipitate as the insoluble mercuric sulfide.

E. Levels in Media

The absolute concentrations of total mercury differ by orders of magnitude in different environmental media. As stated above, atmospheric levels distant from known sources of pollution are in the range of 1-2 ng per cubic metre. Levels in sea water, estimated as 0.5-3.0 ng Hg/litre (*WHO, 1989*), are roughly 1000 times higher, and levels in soils are highest of all being about one million-fold the water levels, measured in units of mg/kg.

The successful speciation of mercury at the low levels found in air and water was one of the triumphs of modern environmental science. The speciation in air has already been discussed. Sea water has been shown to contain all the major environmental forms of mercury – Hg^{o}, Hg^{++}, CH_3Hg^{+} and $CH_3Hg\ CH_3$. Due to the high concentration of the chloride anion, Cl^{-}, Hg^{++} in sea water forms several chloride complexes, $HgCl^{+}$, $HgCl_2$, $HgCl_3^{-}$ and $HgCl_4^{=}$. The uncharged $HgCl_2$ complex is believed to be the species of inorganic mercury that can diffuse across biological membranes, as well as methylmercury.

Mercury levels in environmental media, per se, are not important direct contributors to human exposure and uptake of mercury. The levels of total mercury in water and air are so low that human intake via inhalation and drinking water is negligible. Atmospheric concentrations of mercury levels can rise to levels in enclosed spaces, such as in mercury-related occupations where inhalation may become an important route of entry. Mercury vapour released from amalgam tooth fillings in the mouth is believed to be the predominant exposure to this form of mercury for the general population. In most cases, blood or urine levels resulting from amalgam exposure are well below toxic levels as defined by a WHO expert group (WHO, 1991).

F. Biotransformation and Bioaccumulation in Fish

Swedish environmental scientists, in the late 1960s, discovered that mercury could be methylated by micro-organisms resident in aquatic sediments (Jensen and Jernelov, 1969). No matter what the chemical form of the mercury discharged into rivers and lakes, it was first transformed to Hg^{++} which, in turn, became the substrate of methylation reactions in microbes to produce mainly the monomethylated mercury and possibly some dimethylmercury. The organisms responsible for the biotransformation, the methanogenic bacteria, are some of the oldest known life forms on the evolutionary scale. Thus, the realization dawned that methylmercury was not just a recent product of the chemical industry but a natural product present since life first started on this planet.

Methylmercury leaves the micro-organisms to enter an aquatic food chain that, step by step, magnifies the concentration of methylmercury in living tissues. From micro-organism to plankton to herbivorous and finally to the carnivorous fish at the top of the food chain, the concentration may be increased a million-fold from less than 1 ng/litre in water to 1 mg/kg or more in carnivorous fish such as pike, walleye and bass in freshwater (Lowe et al., 1985; Bahnick et al., 1994) and shark, swordfish and fish-eating sea mammals in ocean waters (National Marine Fisheries Service, 1992).

Virtually all the total mercury in fish muscle is in the form of methylmercury attached to the muscle protein. The actual concentration depends on the species of fish (i.e. the stage of the food chain). For example, fish at the bottom of the chain may have levels as low as 0.01 mg/kg versus large fish at the upper end having levels of over 1 mg/kg, a range of at least a hundred fold. The mercury levels also depend upon the age (length is often used as a surrogate for age) as

methylmercury accumulates during the life of the fish.

Levels of methylmercury in freshwater fish, usually lake fish, are determined by what are described as "within lake factors." These include properties of the lake water such as the pH, temperature, sulfate levels and dissolved organic carbon concentration and degree of oxygenation, as well as characteristics of the lake itself such as the drainage or catchment areas of the lake and the presence of wetland in the drainage areas. Wetlands characteristically are rich in methylmercury production. The combination and permutation of those "within lake factors" make it impossible to identify a "generic" lake from the point of view of methylmercury production. Each lake and freshwater system is unique.

Acidification of bodies of fresh water from industrial discharge of acidic gases also leads to elevated levels of methylmercury in fish. It has been proposed that a low pH enhances the overall methyl process (Winfrey and Rudd, 1990).

The impoundment of bodies of natural water for hydroelectric production has also led to the elevation of methylmercury levels in fish in several provinces. Elevated levels persist for many years after impoundment (Ramsey and Ramial, 1986). The mechanisms underlying the mobilization of mercury are not completely understood, but it is suspected that one factor is an increase in the rate of methylation of inorganic mercury naturally present in sediments (Hecky et al., 1986).

An important environmental correlative is that fish levels of methylmercury are not connected to deposition rates of mercury in lakes. Local factors such as acidification, impoundment, the catchment area of a lake and other factors discussed above dominate the availability of methylmercury in the aquatic food chain. In fact, no quantitative connection has yet been made between anthropogenic sources to the atmosphere and methylmercury levels in fresh and ocean water fish.

G. Human exposures to Mercury

As a result of the cycling of mercury from both anthropogenic and natural sources, humans are exposed in various ways (Table 1). Daily intake of all forms of mercury is mainly from food. Within the food categories, fish and shellfish are the predominant source. It is possible that mercury in certain other foods, such as dairy products, meat, poultry and eggs, may derive ultimately from

fish as fish meal is used in animal livestock fodder.

Table 1. The estimated average daily intake to total mercury for Canadian Great Lakes adult residents (adapted from Health Canada, 1998)

	Estimated intake[1] µg Hg/day		
Medium	Total	inorganic[2]	methyl
Air indoor	0.70	0.70	
outdoor	<0.07	0.07	
Soil & house dust	<0.07	<0.07	
Drinking water	<0.07	<0.07	
Food	4.10	4.10	
dairy products	0.19	0.19	
Meat/poultry/eggs	0.17	0.17	2.30[3]
fish/shellfish	2.60	0.30	
non-alcoholic drinks	0.40	0.40	
all other foods	0.74	0.74	
total food	4.10	1.80	2.30
Average for adults, all media	4.90	2.60	2.30

(1) The intake numbers are probably higher than the actual amount absorbed into the body (see text).

(2) Includes inorganic compounds in food and water and mercury vapour in inhaled air. It is assumed that virtually all mercury in media other than fish is inorganic.

(3) Assuming 90% present in edible tissues is as methylmercury and 10% as inorganic mercury.

The contribution of inorganic and methylmercury to total mercury intakes have been estimated in Table 1. All mercury in media other than fish and shellfish has been assumed to be in the inorganic form, based mainly on current knowledge of the mercury cycle. A small fraction of

[1] According to the model of disposition of methylmercury in the body as discussed later in this document, long-term, one year or more, daily intake of methylmercury will lead to a blood level of 0.64 x 2.3 ppb = 1.5 ppb. The concentration of hair to blood in Canada adopted in the first volume of *Methylmercury in Canada* is 300:1. Thus, the corresponding hair level is 1.5 x 300 = 450 ppb or 0.45 ppm.

atmospheric mercury is known to be in the methylated form. It is also possible that a small fraction of methylmercury may be in animal products. However, from what is known of the metabolism of methylmercury this organic fraction can be expected to be small (see section on the disposition of methylmercury in the body).

The figure of 2.3 μg Hg/day as methylmercury agrees well with a WHO estimate of 2.4 μg Hg/day for the general population (WHO, 1990). The Canadian figure for intake of inorganic mercury, 2.6 μg Hg/day, is about 40% lower than the WH0 estimate. The difference probably lies within the error range as data on the species of mercury in foods other than fish is sparse so that estimates have a correspondingly large error. Whereas most of the methylmercury intake will be absorbed into the body, only a small fraction of inorganic mercury in the diet, about 7%, will be absorbed.

Methylmercury in fish is thus the predominant form of mercury in the body. This form of mercury is taken up from the bloodstream into scalp hair. Thus, a general correlation is seen between methylmercury intake estimated from dietary surveys and the corresponding hair levels (Airey, 1983). A long-term daily intake of 2.3 μg Hg as estimated for residents of the Great Lakes would correspond to a blood level of 1.5 ppb and to a hair level of 0.45 ppm. The latter is good agreement with observed levels in hair of North American adults of 0.36 ppm, range 0.05-0.6 (Smith et al., 1997). These figures indicate that residents of the Great Lakes Basin are not experiencing an unusually high intake of methylmercury.

Of special concern in Canada are those communities that depend on freshwater fish and seafood as a major component of their diet. The previous volumes of *Methylmercury in Canada* and the current volume document levels in blood and hair in First Nations and Inuit communities far in excess of those found in the general population. The source of mercury, the seasonal patterns of intake and the yearly trends in mercury levels have been discussed in previous sections of this report. Clearly, such elevated levels require a thorough examination of health risks from methyl and inorganic mercury.

HUMAN HEALTH RISKS FROM METHYLMERCURY IN THE DIET

This section will consider the disposition of methylmercury in the body, its adverse health effects and finally the risks of adverse effects as they relate to levels of methylmercury in blood and hair.

DISPOSITION OF METHYLMERCURY IN THE BODY

The environmental cycling of mercury results in dietary intake of methylmercury. Environmental exposure to other forms of mercury (mercury vapour and the inorganic compounds) are insignificant. This section therefore will focus on the metabolic rate of methylmercury in human diet.

A. Tissue Distribution

Studies on human volunteers given tracer doses of radioactive methylmercury (Aberg et al., 1969; Miettinen, 1973) reveal that virtually all methylmercury in the diet (95%) is absorbed into the blood stream. From the blood stream, methylmercury distributes to all tissues in the body. The distribution process is completed in about three days (Kershaw et al., 1980). About 5% of the body burden is found in the blood compartment with the rest of the methylmercury attached to the red blood cells. The red blood concentration is some 20 times higher than the plasma level. Methylmercury crosses the blood brain barrier resulting in concentrations in the brain approximately 5 times the level in whole blood. It crosses the placenta. Levels in infant (cord) blood are about twice those in maternal blood due to the increased binding to red cells. Methylmercury distributes throughout the fetal tissue.

Data are scarce on concentrations of methylmercury in the human fetal brain. A recent report (Cernichiari et al., 1995) indicated that the average concentration of total mercury in autopsy brain of neonates correlated with concentrations of mercury in maternal scalp hair at the time of delivery. On average, the maternal hair levels was about 15 times higher than corresponding levels in the fetal brain. In the non-pregnant adult, studies reviewed by a WHO expert group (WHO, 1990) suggest that the hair level is some 40 times higher than the brain level. These estimates suggest that the fetal brain may be some two to three times higher than the corresponding level in the maternal brain. Animal

data (for review see Clarkson, 1992) also indicate the fetal brain level is higher than maternal brain by about a factor of two.

B. Metabolism to Inorganic Mercury

Methylmercury can be metabolized to inorganic mercury by cleavage of the carbon-mercury bond. Phagocytic cells, present in most tissues, are known to perform this function (Suda and Takahashi, 1986). Inorganic mercury is preferentially accumulated in kidney tissue. Thus, kidney levels of inorganic mercury tend to build up after long-term exposure to methylmercury. This may be one explanation of the high proportion of inorganic mercury found in urine samples in people exposed to methylmercury.

Methylmercury is also converted to inorganic mercury in the brain. Thus, inorganic mercury also builds up in this tissue as it has a limited ability to cross the blood brain barrier. Elevated concentrations of inorganic mercury have been found in autopsy brain samples from people who have died many years after an acute exposure to methylmercury (Davis et al., 1994). The inorganic mercury is believed to be in an inert, insoluble form that can remain in brain tissue for many years, perhaps for the lifetime of the individual (WHO, 1990).

C. Pathways of Excretion

Methylmercury is excreted in adults by a complex process involving the liver, bile and intestinal tract (for a recent review see Ballatori, 1994). Methylmercury in liver cells is secreted into bile whereupon it is partially reabsorbed back into the blood stream via the gall bladder and intestinal tract. The remaining methylmercury from bile is broken down to inorganic mercury by the microflora in the intestine. The inorganic form of mercury is poorly absorbed (about 7% of an oral dose is absorbed) across the intestinal wall resulting in the majority being excreted in the feces. Methylmercury attached to exfoliated intestinal cells may also contribute to the pool of mercury that is demethylated in the intestinal tract.

Methylmercury is not secreted in bile by suckling animals (Ballatori and Clarkson, 1982) and the microbial demethylation process is virtually absent (Rowland et al., 1983). The biliary secretion process starts abruptly at the end of the suckling period. No information is available on human infants but the animal data suggest that excretion of methylmercury may be much slower than in adults.

D. Biological Half-times

The rate of excretion can be expressed quantitatively in terms of biological half-times. In the case of methylmercury, its elimination from the body can be approximated by a single biological half-time, that is to say, the time taken for the amount in the body to fall by a half. The first studies used methylmercury labelled with radioactive mercury (Aberg et al., 1969; Miettinen, 1973). They observed that a half-time in the whole body of about 70 days corresponds to a rate of excretion of 1% per day. The drawback in using radioactive mercury is that the radioactivity measures not only methylmercury but also the inorganic mercury split off from the parent compound. The true half-time of methylmercury is probably shorter than 70 days.

The half-time in blood has been reported to average about 44 days (range 32 to 60 days) in the most recent paper (Smith and Farris, 1996). In this case, methylmercury was distinguished from inorganic mercury. The half-time in blood is the important parameter underlying the relationship between daily intake of methylmercury versus blood concentrations (see below).

Half-times have also been calculated from longitudinal analysis of samples of scalp hair. Concentrations of methylmercury in newly formed hair are proportional to the simultaneous blood levels (Amin-Zaki et al., 1976; Kershaw et al., 1980; Hislop et al., 1983). Hair grows at the average rate of slightly over 1 cm per month. Thus, measuring mercury levels in consecutive centimetres will give a month-by-month recapitulation of mercury levels in blood and has been used to estimate half-times after the cessation of exposure to methylmercury (Amin-Zaki et al., 1976). Some estimates have been made in studies of populations that have received an acute exposure to methylmercury (e.g. Cox et al., 1989) and in volunteer subjects who had received measured doses of methylmercury (Kershaw et al., 1980). In general, hair analyses tend to give a much wider range of individual half-times than those estimated from blood measurements – the first report gave a range of 30 to 189 days (Al-Shahristani and Shihab, 1974). A variety of errors and uncertainties could account for the wide range (e.g. different growth rates of hair, misalignment of individual hair strands and perhaps analytical errors). The measurement of carefully timed blood samples gives a more accurate picture.

E. Mechanisms of Transport in Tissues

Methylmercury chloride is the compound of methylmercury most commonly used in experimental work. It happens to be lipid soluble and thus gives the impression that methylmercury is absorbed and distributed in the body in a lipid soluble form. This is not the case. As discussed in the introduction, the methylmercury cation rapidly attaches to the sulfydryl groups that are primarily found in water-soluble molecules such as proteins and amino acids. Current research suggests that methylmercury attaches to the sulfydryl ligand in the amino acid, cysteine (Kerper et al., 1996). The resulting complex mimics the structure of the essential amino acid, methionine and thereby gains entry into cells on the protein carrier for this amino acid. Once inside the cell, methylmercury switches its association to the sulfydryl peptide, glutathione, present in most cells at high (millimolar) concentrations. Current research also suggests that methylmercury is exported from cells (e.g. from liver cells into bile) as a complex with glutathione (Ballatori, 1994). It is of interest that when biliary secretion of methylmercury switches on at the end of suckling, so does the biliary secretion of glutathione (Ballatori and Clarkson, 1982).

F. Blood Levels After Long-term Intake

The uptake, distribution and excretion of methylmercury in humans can be described mathematically leading to a quantitative relationship between dietary intake and levels of methylmercury in indicator media such as blood and scalp hair (WHO, 1990). The relationship between daily long-term intake and blood levels of methylmercury is important to estimating human health risks from methylmercury in the diet, since the risk of developing signs and symptoms of methylmercury poisoning is usually related to blood (or hair as surrogate for blood) levels.

The daily intake of methylmercury will lead to a rise in the amount in the body until a steady state level is attained when intake is balanced by excretion. The time required to reach this steady state is a period of time equivalent to five half-times. For example, if the blood half time were 44 days, it would take about 230 days or close to 7 months' exposure to attain its maximum steady state level.

The equation relating blood concentration (C μg/l) to daily intake (d μg/day) is given by

$$C = \frac{d.A.f}{V\ (\text{litres}) \times b\ (\text{day}^{-1})} \quad \ldots\ldots (1)$$

where d (μg/day) is the daily intake, A is the fraction absorbed, and f is the fraction of the dose present in blood, V (litres) the volume of blood and b the kinetic rate constant. The latter is related to the half-time in blood T ½ by the expression

$$b = \ln 2/T1/2 \quad \ldots\ldots (2)$$

Substituting the appropriate values into equations (1) and (2), we obtain a quantitative relationship between daily intake and blood level for people experiencing long-term exposures to methylmercury:

$$C = \frac{0.05\ T\,½}{5 \times \ln 2} = \frac{0.05}{5} \cdot \frac{44}{0.69}$$

$$= 0.64 \cdot d \quad \ldots\ldots (3)$$

Clearly, the relationship between intake and blood level will depend on what values are assumed for the variables in equations (1) and (2). A, assumed to be 95%, although animal experiments suggest it may be affected by dietary factors, V (in litres) is assumed to be 7% of the body weight (in kg) and is clearly dependent upon age. Most studies are consistent with f in the range of 0.05 to 0.07. The reported range in blood half-times (T ½) is large if we include half-times calculated from hair analyses. However, if we accept only those half-times calculated for analysis of blood samples, mean values for each study falls in a much narrower range, 44 to 53 days.

The first observational test of the relationship was made by a Swedish Expert Group in the late 1960s which found that a daily intake of 300 μg corresponded to a blood level of 200 μg/L. This would correspond to a multiple of d of 0.66 and compares well with the theoretical value of 0.64 in equation (3) based on the latest published information (Smith and Farris, 1996) in blood half-times.

ADVERSE HEALTH EFFECTS OF METHYLMERCURY

A Swedish Expert Group in 1971 was the first to publish a comprehensive treatise on the human toxicology and risk of adverse effects from exposure to methylmercury in fish (Swedish Expert Group, 1971). Since then, numerous papers have added a considerable amount of detailed knowledge but the general descriptions and conclusions of that group remain essentially unchanged to this day.

This section will first deal with the major toxic effects with special emphasis on clinical and pathological observations in human victims of poisoning. The next section will be an evaluation of the literature as to the risks of poisoning from dietary intake.

A. The Clinical Pathology of Methylmercury

Studies of several outbreaks of mass poisoning augmented by a number of case reports have provided a detailed picture of the clinical signs and symptoms and the major pathological changes in the target organ, the brain (WHO, 1990). A primary distinction must be made between the effects on the mature brain versus those on the developing brain, especially during the prenatal period.

1. Adult Exposure

a. Effects on the Nervous System

The signs and symptoms of methylmercury poisoning derive mainly, if not entirely, from damage to the brain. The first symptom reported is paresthesia, a numbness or tingling sensation in the extremities of the hands and feet and circum-orally. More advanced poisoning is indicated by ataxia, constricted visual fields, dysarthria and deafness.

These signs and symptoms result from focal destruction of neuronal cells in specific areas of the brain. For example, the loss of coordination typified by cerebellar ataxia is assumed to be due to the destruction of the granule cells in the cerebellum. The damage appears to be highly selective to the small granule cell as neighbouring Purkinje cells are only slightly affected, if at all. Constriction of the visual field results from the loss of neurons in the visual (Calcarine) cortex (Hunter and Russell, 1954). Thus, the most recent evolutionary development of the brain, the neo-

cortex, appears to be the main target with the cerebellum being an important but perhaps less sensitive target. The deeper, more primitive areas of the brain such as the limbic system and brain-stem – the site of control of vital functions such as heart beat and respiration – do not appear to be affected. Death from methylmercury poisoning is most likely due to secondary infections and complications arising from primary damage to the higher functions.

The selectivity to the higher regions of the brain may explain why organs that are evolutionary less developed are less sensitive. Fish and other aquatic organisms require much higher doses compared to mammals before they succumb to the toxic effects of methylmercury. The first outbreak of mass poisoning took place in Minamata in the 1950s when heavily contaminated fish were consumed (for a review, see Hunter, 1969). Amounts of methylmercury that ultimately would prove toxic to the fish consumers continued to accumulate up the aquatic chain without damage to the intermediate species in the chain. Cats and humans were the first to show toxic effects. Hearsay evidence for the epidemic indicates that only fish having massive tissue levels indicated signs of swimming difficulties.

The damage to the brain is irreversible as neuronal cells are destroyed. In the mature brain, these cells cannot be replaced (Hunter and Russell, 1954; Takeuchi, 1961). The remarkable selectivity to the brain is puzzling as methylmercury distributes to all tissues in the body. The brain levels are actually lower than some tissues such as liver which are not damaged. The reason for the focal damage to specific areas in the brain is likewise a mystery despite almost 30 years of research. One early study suggested that all cells in the neo-cortex and cerebellum suffered initial damage but certain cell types were able to repair the damage (Syversen, 1977). Thus, the granule cells in the cerebellum lacked the repair capability of the large Purkinje cells.

The primary biochemical process inhibited by methylmercury has not been identified. Several papers suggest that some of the steps in protein synthesis are among the first to be affected (Yoshino et al., 1966; Syversen, 1977). Other biochemical sequelae have been described, such as lipid peroxidation (Verity and Sarafian, 1991) and DNA damage (Costa et al., 1991). Perhaps the processes of damage repair are of greatest interest as they may ultimately decide which cells will survive and which will succumb. Surprisingly little research has been devoted to repair mechanisms.

An important consequence of the pathology of methylmercury poisoning is that the brain damage is irreversible. Thus, it is logical to assume that it will be the maximum level ever attained

in the brain that will be responsible for the full extent of brain damage and for the ensuing signs and symptoms. Studies of a mass outbreak from an acute exposure confirms that the peak level is associated with the risk of poisoning (Bakir et al., 1973).

This greatly complicates epidemiological studies of human populations who are exposed for a lifetime to methylmercury in the diet. A cross-sectional or prospective study will reveal only current or future mercury levels whereas the signs and symptoms may have been caused by prior exposure from a much earlier period.

Another important aspect of human methylmercury poisoning is the presence of a long latent period after cessation of exposure. In the Iraq outbreak where a high intake took place over a period of weeks, the first signs and symptoms did not appear for some 26 to 38 days after exposure had stopped (Bakir et al., 1973). The reason for this long latent period is unknown. It adds to the hazardous properties of this form of mercury as a victim may consume what will eventually prove to be a severely toxic dose without experiencing any warning signs during the exposure period and for some time afterwards.

b. Other Effects

The selective damage to the central nervous system was demonstrated in studies of the Minamata and Iraq outbreak. Polyneuritis may be present in severely affected patients (for a review see Kurland, 1973). Other tissues in the body are not affected even in severe cases of poisoning. No evidence has been reported that the reproductive function is impaired in humans despite some suggestive evidence from animal studies (Khera, 1973).

Studies on a small group of fish eaters in Sweden raised the possibility that methylmercury damaged chromosomes (Skerfving et al., 1974). Animal studies and tissue culture experiments suggest that methylmercury may be a weak mutagen (Ramel, 1972).

2. Prenatal Exposure

Outbreaks in Japan documented that children may suffer mental retardation (cerebral palsy) when mothers are heavily exposed to methylmercury during pregnancy (Kurland, 1973). At lower maternal exposures, less severe effects were seen in Iraq, such as delayed achievement of developmental milestones (age of first walking and talking) and the presence of abnormal reflexes

(Marsh et al., 1987). Epidemiological studies on populations having elevated levels of methylmercury from fish consumption have found limited evidence of diminished neuropsychological performance, although no firm conclusions can be drawn at this time (see dose-response section).

The pathology of the brain of severe cases of prenatal poisoning reveals that methylmercury damages the basic cellular processes of brain development – namely, the growth and proliferation of nerve cells and the process of migration of the cells from their origin in the neuronal crest to their ultimate anatomical destination (Choi et al., 1978). These inhibitory effects on cell division and migration probably result from a common biochemical lesion. Experimental studies have revealed that the first effect seen at the lowest concentrations of methylmercury is the inhibition of the assembly of microtubles (Miura and Imura, 1991). Microtubles are formed by a tread milling process – being assembled at one end of the tubule, and disassembled at the other end. Thus, the inhibition of the assembly process still allows the disassembly to continue until the tubule disappears. Microtubles are a part of the internal structure of the cell and are essential for the processes of cell division and migration.

The fact that methylmercury inhibits the most basic mechanisms underlying brain development results in a widespread diffuse pathology, as distinguished from the focal damage in the adult and mature brain. Thus, the small head size seen in cases of severe poisoning is no doubt a result of the inhibition of cell division. Likewise, the inhibition of neuronal migration accounts for the presence of atopic cells that have failed to travel to the appropriate anatomical area of the brain.

RISKS OF ADVERSE HEALTH EFFECTS

The key to estimating the risks of adverse health effects from ingestion of methylmercury in fish is the so-called dose-response relationship. Such relationships are routinely established in animals. In these toxicity tests, animals are allocated to a number of well-defined dosage groups whereby the percentage of poisoned animals in each group can be graphically compared with the dose. The "lowest observed adverse effect level" (LOAEL) is the lowest dose group where a statistically significant effect is seen. The so-called "no observed adverse effect level" (NOAEL) is the next lower dose where no statistically significant effect is seen. Such studies in animals are used in toxicological tests on chemicals to establish safe exposure for humans.

However, such toxicological tests in animals have limited relevance in the case of methylmercury due to the many difficulties and uncertainties in extrapolating results to humans. Methylmercury at the lowest toxic levels may produce subtle neurophysiological changes that would be difficult to detect in animals. The disposition of methylmercury in animal tissues differs from that in humans (WHO, 1990). For example, variable and sometimes large amounts (up to 50% of the dose) of methylmercury is accumulated by animal fur, thus making it impossible to compare ingested dose in animals and humans. Blood to brain ratios differ. The blood to brain ratio in the rat differs from that in humans by about two orders of magnitude. Few studies have reported brain levels that should allow more reliable extrapolation to humans. However, animal experiments are useful in studies directed toward elucidating mechanisms of action of methylmercury.

For these reasons, estimates of human health risks are based on studies of exposed human populations. Published studies are on populations where overt cases of poisoning have occurred (e.g. Japan and Iraq) or on populations having elevated methylmercury levels from consumption of fish or sea mammals. Some have been retrospective (e.g. Japan and Iraq); others have been cross-sectional or prospective epidemiological investigations (e.g. fish-eating populations). In all cases, methylmercury levels in blood and scalp hair have been used as a measure of the dose (e.g. as surrogates for levels in the target organ, the brain). Total mercury is usually measured as methylmercury and accounts for most of the mercury (95% in blood samples and over 80% in hair samples). The end points used as an indicator of methylmercury toxicity have ranged from overt clinical signs and symptoms to statistical differences in scores of neuropsychological tests. As in the

previous section, distinction is made between prenatal and adult exposures.

A. Risks from Adult Exposure

The first comprehensive review to estimate human health risks was made by a Swedish Expert Group in the late 1960s and published in 1971. They relied on the two outbreaks in Japan (especially the outbreak in Niigata in 1965) to establish the lowest blood and hair levels associated with adverse effects in adults. They concluded that the LOAELs in blood were 200 µg Hg/L and 50 µg/g in hair. No statistical evaluation of actual risks was possible but they assumed a figure of 5% as the LOAEL, as this is the practical limit of detection in such studies. A subsequent review by the WHO Expert Group (WHO, 1972) adopted the Swedish conclusions on the lowest effect levels. Both the Swedish and WHO Expert Groups applied a "safety factor" of 10 to derive maximal tolerable levels, namely, 20 µg Hg/L in whole blood and 5 µg Hg/g in hair.[2] The safety factor was applied to allow for a range of susceptibility in the general population, including the presumed greater sensitivity of the developing brain (Swedish Expert Group, 1971; Friberg, 1973). After the passage of almost 30 years and the publication of new epidemiological studies (see below), these conclusions have withstood the test of time.

The outbreak in Iraq gave remarkable confirmation of the Swedish-derived human LOAELs. In this case, the body burden associated with the onset of the first symptoms of poisoning in adults was estimated to be within the range of 25- 40 mg Hg as methylmercury (Bakir et al., 1973). Using conversion factors for distribution of methylmercury in the body (see the previous section on "Disposition"), these figures convert to blood levels of 250- 400 µg/L or 60-100 µg/g in hair. Given the uncertainties in numbers derived from epidemiological investigation, the Iraqi numbers are in excellent agreement with the Swedish LOAELs derived by the Swedish Expert Group for the observations in Japan.

All subsequent studies have been on fish-consuming populations where dietary intake of methylmercury presumably was lifelong. This presents a special problem as the peak level of methylmercury may have been experienced before the studies took place.

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[2]The hair level of 6 µg Hg/g adopted by MSB (see Figure 1, page 3) came about because a hair to blood concentration ratio of 300:1 was used instead of the 250:1 ratio used by the two expert groups.

Adult studies, such as they are, have failed to find effects due to mercury at blood or hair levels below the NOAEL derived by the Swedish and WHO Expert groups. A study was conducted by the McGill University in the 1970s of adult Cree Indians with elevated blood and hair levels due to consumption of methylmercury in fish (McKeown-Eyssen and Ruedy, 1983). Some fish were specially contaminated due to local pollution by mercury released from paper pulp plants. Although neurological signs and symptoms were found, mercury could not be established as the cause. The main contributing factor was the age of the subjects. Several other factors in this population could also contribute to the observed signs and symptoms. In this study, mercury levels at the time of the study exceeded the LOAEL levels of 200 μg/L in blood and 50 μg/g in hair in a number of subjects. The McGill study is the only published large-scale study in Canada, and the only one on consumers of fresh water fish.

Spitzer et al. (1988) reported an epidemiological investigation of 321 adult Crees and Algonquins residents in northwest Quebec which was conducted to ascertain the health impact from a local industrial source of mercury pollution. Mercury contaminated a freshwater system, resulting in elevated levels of methylmercury in fish and consequent human exposure.

Several approaches were applied to assess mercury exposure in the study population: 1) the concentration of total mercury in hair (the average for the entire length of the hair sample, the first 9 cm from the scalp and segment greater than 20 cm from the scalp; 2) levels in whole blood; and 3) fish consumption.

Only two hair samples were reported to have mercury levels in excess of 40 ppm, four samples were greater than 20 ppm average for the whole length, five for the 9-cm segments and 28 of the greater than 20-cm segments. The correlation coefficient between hair and blood was 0.81, but the hair segment used in this correlation was not stated.

Eleven health outcomes were measured as indicators of neurological damage and general health status of the subjects. Multiple linear regression analysis was used to assess the relationship between health outcomes and hair levels of methylmercury. The analysis took into account a number of potential confounders, including age, sex, diabetes and alcoholism.

Statistically significant correlations ($p < 0.05$) with one or other or all of the three measures of hair mercury were found for four health outcomes: 1) any neurological or opthalmological abnormality, 2) coexistent upper and lower extremity tremor, 3) abnormalities recorded by the data-

gathering technician and 4) abnormal sensory nerve conduction velocities. The brief discretion of these health endpoints does not seem to exclude the possibly of overlap; that is, a specific abnormal measure in one category is also responsible for an abnormal score in another category.

This population, as do other freshwater fish-consuming First Nations people living in northern Canada, experiences marked seasonal changes in hair mercury levels. The highest level is achieved in late summer and early fall when weather conditions prevent further fishing. The lowest levels occur just before next year's fishing season (Phelps et al., 1980). Seasonal changes are indicated in this population by differences in average mercury levels in the 9 cm versus the greater than 20-cm hair segments. Even the longest hair samples rarely allow recapitulation of exposure for more than two years. Thus, we have no information on the earlier exposure of these subjects.

Longitudinal analysis was not carried out so we have no information on peak concentrations in the various hair segments used in this study. The average levels reported in this paper, especially for long hair segment, should be appreciably lower than the monthly peak.

The lack of information on lifetime exposure and on peak levels precludes the use of this study for risk assessment. However, it is the first study to demonstrate mercury-related health effects in adults from consumption of contaminated fish outside the Japanese incidents and the first to show such effects from mercury that has been methylated in the environment by natural processes.

Lebel et al., (1995) reported the results of a study on 29 young adults exposed to methylmercury in fresh water fish in the Amazon basin. The hair levels of total mercury were in the range of 5.6 to 38.4 μg Hg/g. Results of visual testing showed that colour discrimination capacity decreased with increasing mercury in hair and peripheral field profiles were reduced for those with the highest mercury levels. For women, manual dexterity decreased with increasing hair mercury.

Lebel et al., (1998) reported on a larger group (n=91) from the same area residing in one of the two villages involved in the first report. Only adults were examined. Hair was used as the biological monitor. Longitudinal analyses were performed on consecutive 1-cm segments. In the case of some female participants, the hair segment's total length extended to 24 cm. Four measures of mercury exposure were used: average total mercury, the highest 1-cm segment (peak value), total mercury and methylmercury in the 1-cm segment next to the scalp.

Nine "neurofunctional" endpoints were used, including manual dexterity and visual contrast sensitivity at different frequencies, measured outdoors in shaded areas to control the luminance.

Multiple regression analysis was used to correlate these endpoints with the four measures of exposure, giving a minimum of 36 correlations. Contrast sensitivity at the higher frequencies of 6, 12 and 18 cps gave statistically significant correlations with peak hair levels and with some of the other measures of dose.

The authors state that age adjustments were made, but no details were given. The age range was broad, 15 to 81 years. Stepwise regression analysis was used to assess the influence of confounders on the neurofunctional outcomes, but no explicit list of confounders was given. Socio-demographic data are given that indicate that 7% had worked in mercury mining (presumably exposed to mercury vapour), 34% had a history of malaria and 17% a history of parasitosis. The overlap between these groups was not given.

Also, an extensive number of "clinical" endpoints were measured on a randomly chosen subset of 59. Most clinical parameters were normal. However, a high prevalence of disorganized movement, restricted visual fields and hyperflexia were noted. Mercury-related (mainly peak levels) effects were seen for both movement and visual fields, but not for reflexes.

All members of the study group had mercury levels that never exceeded 50 ppm, the hair level that is currently regarded as the lowest toxic level in adults. The authors point to the "particular physiological importance" of peak levels of mercury. This makes sense as methylmercury, at least in terms of its overt clinical effects, is believed to be an irreversible poison because it destroys neuronal cells (Hunter and Russell, 1954). Thus, the history of exposure may be key to determining the outcome. When did the peak levels occur in this adult population? The longest hair sample could recapitulate exposure over no more than two years. The authors themselves suggest that the adverse effects found in the younger group, those 15-20 years, may be due to prenatal exposure, as mercury releases to the environment due to gold mining started about 20 years ago.

The study raises a toxicological puzzle. If peak values occurring long before this study are responsible for the observed "adverse" effects, why do some of these outcomes correlate with current (within the last two years) mercury levels. Do current peak levels correlate with past levels? Do levels in the mother during pregnancy correlate with levels in the offspring over 15 years later? Alternatively, are the subclinical effects noted here reversible and therefore determined by current levels? Such questions point to the importance of longitudinal evaluations starting with prenatal exposures and continuing to adulthood. Without answers to these questions, we cannot conclude that

this study proves that adult exposures below 50 ppm are associated with adverse effects.

A single cross-sectional evaluation on any adult or even on any prenatally exposed population leaves these key toxicological questions unanswered, makes risk assessment difficult, and the lowest effect levels a matter of conjecture. Unfortunately, all studies on chronically exposed adult populations have been cross-sectional. These include ocean fish-eating populations in Peru, American Samoa, the Mediterranean (WHO, 1990), as well as the Canadian studies reviewed above. Effects such as paresthesia have been found, for example in Peru, but they did not correlate with current mercury levels. Without longitudinal studies, we cannot exclude the possibility that earlier exposure to mercury was responsible.

Thus, we must still rely on the two Japanese outbreaks and Iraq because the limited time of exposure allowed estimates of the true maximum blood and hair levels. The conclusion of the Swedish Expert Group and WHO remain our best evaluation of adult risks, namely that adverse effects from adult exposure should not occur below hair and blood levels of 50 ppm and 200 ppb, respectively.

B. Risks from Prenatal Exposure

Epidemiological studies of prenatal exposures have an advantage over adult exposures in that the dose can be measured over the period of interest – the nine month prenatal period as opposed to the lifetime exposure for adults. Nevertheless, even the prenatal exposures suffer from many complications that make it difficult to establish methylmercury as the cause of any observed adverse effects. The primary argument for an effect of methylmercury is a statistical correlation between methylmercury levels and the prevalence of a given adverse effect. This argument is always open to a scientific challenge, as a correlation, in itself, does not establish causality. This is especially true in the studies to be reviewed where the endpoints (e.g. delayed development) could have numerous causes and where the effects of methylmercury, if anything, will be subtle and difficult to distinguish from the background noise.

A suspicion that the prenatal period may be the most sensitive stage of the human life cycle to methylmercury came from clinical observations on a few infant-mother pairs in the Minamata outbreak in Japan where the prenatally exposed infants appeared to be more severely affected than their mothers (Kurland, 1973). Subsequent animal experiments tended to support these observations

(e.g. Rice and Gilbert, 1990). However, such comparisons are of dubious scientific merit as the effects on the mothers (see Adult Exposure section) are qualitatively different involving different mechanisms than those seen in their off-spring.

A study of 83 infant-mother pairs in the Iraqi outbreak provided the first quantitative dose-response relationship between the peak levels of methylmercury in the mother during pregnancy and the risk of adverse effects in her off-spring (Marsh et al., 1987; Cox et al., 1989). The reported effects were delayed achievement of developmental milestones (first walking and talking) and the presence of abnormal tendon reflexes. Statistical estimates indicate NOAELs in the range of 10 to 20 μg/g peak maternal hair in pregnancy. The statistical uncertainties in these estimates were wide ranging, from as low as 7 μg/g to as high as 180 μg/g depending on the assumptions underlying the statistical models. A recent re-analysis of the same data confirmed the wide range of uncertainty in the estimated NOAELs (Cox et al., 1995; Crump et al., 1995).

Also in the 1970s, an epidemiological study was conducted by the McGill group on prenatal exposures in the same Cree population where adult expressions were studied (McKeown-Eyssen et al., 1983). The mean methylmercury level in maternal hair was 6 μg/g in 247 infant-mother pairs, including a high mercury group with hair levels in the range of 13 to 24 μg/g. Only one endpoint – abnormal reflexes – was more prevalent in the highest mercury group. The endpoint combined both depressed and enhanced reflexes into a single abnormal category. This is not the usual practice in neurology. As the authors themselves point out, the findings are of doubtful clinical significance. The authors also noted that the statistical method could have led to an association by chance. No dose-response relationship could be found over the entire range of mercury values. No other endpoint exhibited any association with maternal mercury. Thus, the weight of evidence indicated that no prenatal effects of mercury were found in this population. A detailed critique that arrived at the same conclusion has recently been published elsewhere (Marsh et al., 1995).

In the 1980s, a study was conducted in New Zealand of prenatally exposed infants. The study began with a dietary survey of 10930 mothers, 1000 of whom reported eating more than three fish meals weekly during pregnancy (Kjellström et al., 1986). Only 73 mothers had hair levels > 6 ppm and 31 of them were located for stage 1 of the study. After adding an equal number of low mercury subjects, the control and exposed children represented 0.6% of the mothers originally surveyed. The 31 infant-mother pairs in the high exposure group (mean maternal hair methylmercury 8.8 μg/g)

were compared with an equal number in a low exposure group (mean methylmercury 1.9 μg/g). Statistically significant differences were seen in the Denver Developmental Screening Test (DDST), indicating developmental delays in the high exposure group. The results of these tests were highly dependent on the age of the child. In this study, the ages at examination ranged from 4-6 years. Other confounders may have contributed to the observed differences in development, such as low birth weight and gestational age.

In stage 2 (Kjellström et al., 1989), 61 of the 73 original infant-mother pairs were compared with three groups of equal size and lower fetal exposure for a total study group of 248 children. The children were subjected to an array of psychological and behavioural tests. Statistical analyses indicated that high prenatal exposure (peak maternal hair approximately 25 μg/g) decreased performance in many of these tests. The contribution of methylmercury to the decrease in test performance was much less than that of other factors, such as ethnic differences. Even when all known or suspected confounding variables were taken into account, only a small fraction of the decrease in performance could be accounted for. It is therefore difficult to build a convincing case that maternal exposure to methylmercury was actually the cause of the relatively small fraction of decreased performance that was statistically associated with maternal hair levels during pregnancy.

Another study on an ocean fish-eating population was also conducted in the 1980s in coastal villages in Peru (Marsh et al., 1995). This involved 131 infant-mother pairs having mean methylmercury levels in pregnancy of 8.3 μg/g with a range of 1.2 to 30 μg/g. The frequency of neurodevelopmental abnormalities did not correlate with maternal exposure to methylmercury. The authors stated that few confounding factors existed in this population as the mothers did not smoke and consumed little or no alcohol. There was no evidence of malnutrition and socio-economic differences were small.

Two major studies are now in progress – one in the Faroe Islands where methylmercury exposure comes primarily from the consumption of pilot whale and the other in the Seychelles Islands where ocean fish is the source. A study in the Faroe Islands on pre and postnatal exposure to methylmercury established a cohort of 1023 infant-mother pairs between March 1986 and the end of 1987 (Grandjean et al., 1992). Umbilical cord blood samples had a median level of total mercury of 24.2 μg Hg/L with 21% in excess of 40 μg Hg/L. Maternal hair concentration was measured as the average level over varying periods of times during pregnancy but all were in excess of 5 cm

(roughly 5 months of growth). Correlation of these average hair levels with cord blood levels was statistically significant but some scatter was apparent. The average hair to blood ratio was 191, somewhat less than most published levels (WHO, 1990). Pilot whale meat was the major dietary source of methylmercury. The average concentration of mercury was quoted as 3.3 μg Hg/g with about 50% present as methylmercury. In contrast, cod fish, the most popular dietary item of fish consumed, has an average methylmercury level of only 0.07 ppm.

Five hundred and twenty-three infants selected as consecutive births from this cohort were followed up for three developmental milestones during the first 12 months (Grandjean et al., 1995). The developmental milestones usually attained between the ages of 5 and 12 months were sitting, creeping and walking. A surprising finding was that early achievement of these milestones was associated with higher mercury levels in the infants' hair. However, early milestone development was also associated with the length of breastfeeding which, in turn, was associated with infant hair mercury levels. No other potential confounders could be found. It was concluded, therefore, that the correlation of development with infant hair mercury was spurious and represented a beneficial effect of breastfeeding. The authors noted that this beneficial effect took place despite high levels of PCBs in maternal milk.

Unlike the studies reported on 30-month-old Iraqi children, maternal hair levels were not associated with delayed achievement of the developmental milestones including the one common milestone, age at walking, measured in both studies. The Seychelles study (see below) also found no correlation.

Dahl et al., (1996) reported on computer-assisted neurobehavioural tests conducted on 917 children at 7 years of age. Three tests from the Neurobehavioural Examination System were administered. Slight but statistically significant decrements were seen with increased levels of prenatal exposure to neurotoxicants, as evidenced by the mercury concentration in cord blood. However, the authors noted that whale blubber contains other neurotoxicants, including PCBs. The latter could not be excluded as the underlying cause of the observed correlation between mercury exposure and neurobehavioural dysfunction.

The most recent report from the study in the Faroes (Grandjean et al., 1997) summarizes the results of all the tests on the 7-year-old cohort of 917. Clinical examination and neurophysiological tests did not reveal any clear-cut abnormalities related to mercury. However, several

neuropsychological tests showed slight but statistically significant negative associations with cord blood mercury levels. Linear regression analysis revealed a statistically significant ($p = 0.05$ or less) negative linkage with mercury in 8 out of 20 such tests. This proportion was increased to 10 out of 20 when other statistical calculations were applied. Cord blood mercury was used as the independent variable. Correlations were seen with maternal and infant hair mercury levels. However, the statistical significance of such correlations disappeared when adjustment was made for cord blood mercury levels.

Linear regression analyses were also conducted after deletion of those children, approximately 15% of the cohort, with maternal hair levels of 10 ppm or higher. Again, cord blood mercury was used as the independent variable. With this restriction, significant p-values were seen in 7 of the 20 tests. In one test, a p-value attained significance ($p<0.02$), whereas with the full group it was non-significant ($p < 0.1$). In two other tests, the p-value attained greater significance than in the full cohort.

Cord tissue concentration for certain PCB congeners were available for 435 of the children in the cohort (the median and range of the cord blood mercury in this group is not given). Four of the psychological tests showed significant negative correlation with the logarithmic transformation of the PCB wet weight concentrations. Nine tests showed significant negative correlations with cord mercury levels (it is not stated whether these are the same tests as in the complete cohort). In the four tests that showed correlations with both mercury and PCBs, the significance of the mercury correlation disappeared in three of the four tests when adjustments for PCBs were made. As the authors point out, PCBs may be an important factor in these tests.

Further analysis of the data from the 7-year examination has been reported (Grandjean et al., 1998). In this analysis, a case-control comparison was made. A case group of 120 children with maternal hair in pregnancy between 10 to 20 μg Hg/g was matched with a control group of 312 children with maternal hair less than 3 μg Hg/g using age, sex, time of examination and the mother's score on the Raven's Progressive matrices as matching criteria. The authors stated that the two groups were almost identical with regard to other factors that might affect neurobehavioural performance. Mild decrements were found in the case group versus the control group in motor function, language and memory.

Despite the fact that this appears to be a well-conducted study on a large group of children

with extensive neuropsychological testing, the interpretation of these findings is not straightforward with regard to effects of methylmercury. This population consumes whale blubber along with the meat. Pilot whale blubber has high concentrations of PCBs and other organochlorine contaminants (Weihe et al., 1996). Attempts were made to use statistical procedures to correct for the deleterious influence of PCBs. However, PCBs were measured in cord tissue on a wet weight basis, a procedure that has not been validated for assessing human exposure. PCBs were measured in only half the cohort. It is not known if correcting for PCBs also "corrects" for any potential adverse influence of the many other contaminants in blubber. It still remains an open question as to what are the relative contributions of methylmercury, PCBs and other contaminants to the mild changes seen in the tests of 7-year-olds.

Although limited evaluations were made on these children at an earlier age (e.g milestones of development), the only substantial evaluation took place at the exam of the 7-year-olds. Without repeated longitudinal evaluation, we have no information on the permanence of the mild effects that were claimed to be associated with mercury. Indeed, the milestone study found beneficial effects correlated with mercury levels as discussed above. Moreover, such mild effects are specially susceptible to confounder influence, as the magnitude of the observed changes is close to background noise. A second evaluation of this cohort is needed before firm conclusions can be drawn.

The latest paper cannot be used to estimate a no effect level. Only summary statistical data are given, such as regression coefficients and p-values. The actual data (e.g. in the form of scatter plots) are not given.

The Seychelles Child Development Study is a prospective investigation started at about the same time as the study in the Faroes. A "pilot" cohort of over 700 infant-mother pairs were recruited in 1987-88. The recruitment of the main study cohort of similar size as the pilot group was started and completed in 1989. The experience with the pilot group was used to guide the experimental design of the main cohort. The off-spring of the mothers in each cohort have been subjected to a range of developmental tests appropriate to the age of the child. In the main study, the children were examined at the same age (within a few weeks) at 6, 19, 30 and 66 months. The study on the children, now 8 years of age, is in progress. The results of the tests on the pilot group up to 66 months of age and on the main cohort up to 30 months of age have been published (Cranmer, 1995; Myers et al., 1997).

The first results from the pilot study indicated that developmental delays, as determined by the DDST, were associated with mean maternal hair levels during pregnancy. However, as in the case of the New Zealand study, the children were not examined at the same age. When the same test was repeated in the main cohort on children at the same age, no correlation with methylmercury was found. The DDST has three outcomes – normal, questionable and abnormal. In both the New Zealand and Seychelles pilot studies, a correlation with maternal methylmercury was seen only when the "questionable" outcomes were included in the "abnormal" category. Combining these categories is not done in the normal clinical application of the test.

Subsequent findings in the pilot and main studies have not yet yielded any convincing evidence that prenatal exposure to methylmercury is causing adverse developmental, behavioural or psychological effects in the Seychelles children. Only in an occasional test was there a suggestion of an adverse outcome (e.g. in the Denver evaluation) in the pilot group which might be attributed to age differences at the time of testing. A further study (Myers et al., 1995) on the pilot group of 217 children at 66 months of age did indicate that the scores of four performance tests (McCarthy General Cognitive Index, Perceptual Performance Subscale, School Language Scale Total Language and Auditory Comprehension Subscale) correlated negatively with prenatal mercury levels. However, the statistical significance of these associations depended on a small number (less than 6 out of a total 217) of outliers and influential points. Follow-up of these children revealed that other factors may have accounted for their abnormal scores.

Myers et al. (1997) described the results of tests designed as a direct comparison of the Iraqi prenatal study. The same developmental milestones were measured, by the same examiner, in children at the same age. No adverse affects attributable to prenatal exposure to methylmercury could be detected in the Seychelles study. As discussed, the original statistical analyses of the Iraqi study (Marsh et al., 1987; Cox et al., 1989) raised the possibility of adverse developmental effects down to maternal hair levels as low as 7 ppm, with a wide margin of uncertainty as confirmed by subsequent analyses (Cox et al., 1995; Crump, 1995). The median hair level in the Seychelles was close to 7 ppm, with the highest level at 26 ppm. Thus, the lower extreme projected from Iraq is not confirmed.

The most recent paper from the Seychelles study (Davidson et al., 1998) reports the results of testing on the main cohort at 66 months of age. Six age-appropriate neurodevelopmental tests were administered: the McCarthy Scales of Children's Abilities; the Preschool Language Scale; the Johnson

Woodcock Applied Problems, and Letter and Word Recognition Achievement Tests; the Bender Gestalt; and Child Behavior Checklist. The outcomes of these tests were compared by multilinear regression analyses to both prenatal (maternal hair mercury) and postnatal (child's hair mercury) measures of exposure to methylmercury.

Scores on the Preschool Language Scale increased with higher levels of pre and postnatal mercury exposures. The Woodcock Johnson test also showed enhanced performance with respect to postnatal exposure levels, indicating a 10% improvement over the full range of exposures. The Bender Gestalt test indicated a substantial reduction in error scores for increasing postnatal exposure of males. The overall reduction in errors was 40%.

Thus, the results of the 66-month evaluation indicated the children were developing well. Evidence was seen of a beneficial factor that covaries with the mercury levels. The authors suggested that this may be due to intake of beneficial constituents of fish, such as the n-3 fatty acids that are known to enhance brain development.

The fact that the two most comprehensive and sophisticated investigations on the effects of methylmercury in fish on child development come to opposite conclusions is not yet explained. The findings of adverse outcomes in the Faroes may be due to a combination of methylmercury with other contaminants, whereas methylmercury appears to be the only contaminant in the Seychelles. Another difference is that whale meat contains methylmercury at average levels about 10 times higher than the average in ocean fish. Thus, a few meals of whale meat might give rise too much higher peak values in pregnancy than an equivalent number of fish meals. Cord blood levels used as the main measure of dose in the Faroes may not reflect peak levels in early pregnancy. Little information is available on this point. A few longitudinal hair analyses have been reported (Grandjean et al., 1992), but they are not sufficient to draw conclusions about the influence of peak levels during pregnancy. Another reason may be the Seychelles has reported on children up to 5.5 years whereas the Faroes examined children at 7 years. The results of the forthcoming tests at 8-9 years in the Seychelles should provide interesting comparison with the Faroes.

Despite the fact that each study in itself does not establish mercury as the cause of any of the observed adverse effects, perhaps all the studies taken together may have some common findings that provide a consistent picture of the influence of prenatal exposure to mercury. In other words, despite findings that most tests in each study indicate no adverse effects due to methylmercury, perhaps one or more comparable tests show up in all the studies consistent with an adverse effect of methylmercury. Unfortunately, the published studies do not contain a sufficiently wide range of different endpoints to test this possibility. The McGill study focuses on neurological functions, whereas the New Zealand study reports on developmental and cognitive tests. It remains to be seen if the Faroe and Seychelles studies share a common outcome.

CONCLUSIONS AND RECOMMENDATIONS

The natural processes of biomethylation and bioaccumulation of environmental mercury, dating back to the earliest evolutionary stages of life on this planet, produce levels in fish at least an order of magnitude lower than maximum levels reported in the Japanese outbreaks. Despite numerous epidemiological investigations looking for subtle changes in the performance of the nervous system, either in adult or prenatally exposed infants, so far have failed to establish that methylmercury in fish causes adverse effects even in populations having unusually high fish consumption.

Table 2 summarizes the results of studies on the prenatal effect of methylmercury. It does not take into account the caveats noted above as to whether there are real adverse effects of methylmercury. The conclusions of the authors are used without qualification. Even using this conservative approach, it may be seen that none of the studies indicates an effect below 5 ppm in maternal hair during pregnancy.

Table 2. Lowest effect levels from prenatal exposure to methylmercury as published in those papers listed in the footnotes

Population	Number	LOEL*	Media/ref.**
Crees	247	13-24 (A)	Freshwater fish (2)
Iraq	83	7-180 (P)	Bread (3)
New Zealand	248	25 (P)	Ocean fish (4)
Peru	131	>8.3 (P)	Ocean fish (5)
Faroes	1023	<10 (A)	Pilot whales (6)
Seychelles	750	13-15 (A)	Ocean fish (7)

* The lowest observed effect level is taken as the lowest observed concentration in hair in pregnancy associated with an adverse development effect or, in the case of the Iraq study, the estimated threshold hair level. These numbers are taken directly from the original publications listed below. As discussed in the text, there are doubts that any adverse effect due to methylmercury was actually detected in these populations with the exception of the high-exposure population in Iraq.

** Mckeown-Eyssen et al., 1983; (3) Cox et al., 1989; (4) Kjellström et al., 1989; (5) Marsh et al., 1995; (6) Grandjean et al., 1997 and 1998; and (7) Davidson et al., 1998.

(A) is the average hair level in pregnancy.

(P) is the peak level during pregnancy.

Some investigators used the highest levels of mercury in pregnancy as the measure of prenatal dose. These peak levels were determined by longitudinal analysis of hair samples. In general, for ocean fish consumers, the peak level is about 50% higher than the average during pregnancy. Thus, the lowest effect levels for the Seychelles should be increased by 50% to be comparable to the results from Iraq and New Zealand.

An important conclusion to be drawn from the studies reported in Table 2 is that they confirm the original evaluation of the Swedish Expert Group and the WHO that adverse effects should not be expected to occur from prenatal exposure to blood levels below 20 μg Hg/L or hair levels below 5 μg Hg/g.

The question arises as to the application of a safety factor to the lowest observed effect levels in Table 2. General speaking, when the most susceptible population is studied, no safety factor is used (Dourson et al., 1992). However, ultimately the size of the safety factor is a socio-political

choice rather than a scientific one. It should reflect the tolerance of risk of the general population, and the cost it is prepared to pay for health protection.

HUMAN HEALTH RISKS FROM INORGANIC MERCURY IN THE DIET

Introduction

The Canadian diet also contains inorganic forms of mercury in addition to methylmercury. This section will summarize what we know about exposure to inorganic mercury in our diet, its disposition in the body, the adverse health effects and an estimate of the health risks. Less space is allocated to this form of mercury because health risks are small, probably non-existent.

Human Exposure to Inorganic Mercury

According to a WHO Expert Committee (WHO, 1991), the diet is the major source of human exposure to inorganic mercury. The average daily intake for adults was estimated as 4.2 μg as compared to 0.002 μg from the air and 0.05 μg from drinking water.

The sources of inorganic mercury in the diet are not known in detail. Most foodstuffs, including fruits and vegetables, have levels of mercury below the analytical detection limit (WHO 1991). Fish contributes about 15% to the total dietary intake. Meat and meat products probably account for most of the remainder. Fish meal is widely used for animal fodder. Methylmercury absorbed from the fish meal is converted to inorganic mercury that is deposited in animal tissues. Kidney and liver probably have the highest levels. The situation in Canada is similar, as summarized in Table 1.

Human breast milk may be an important source of inorganic mercury for infants of mothers having a high intake of fish (WHO, 1991). It has been shown that inorganic mercury may account for up to 80% of total mercury in breast milk of women consuming fish (Skerfving, 1988).

The livers of marine mammals are a special case of high levels of inorganic mercury. Early studies indicated that total mercury levels were as high as 765 ppm (Koeman et al., 1973). Inorganic mercury accounts for a large fraction of total mercury. For example, a recent Canadian study found that inorganic mercury accounts for 88% to 97% of total mercury in arctic marine mammals. The inorganic mercury appears to be present as mercury selenide, an inert insoluble compound

(Wageman et al., 1998). Substantial levels of inorganic mercury are also present in whale meat. For example, pilot whales may have levels in their edible meat tissue in excess of 1 ppm (Juhlshamn et al., 1987). The origin of inorganic mercury in marine mammals is undoubtedly from their diet of fish.

Thus, the aquatic food chain responsible for human exposure to methylmercury is also responsible in most part for our exposure to inorganic mercury. Individuals who have mercury amalgam tooth fillings will also experience exposure to inorganic mercury added to the food during the process of chewing. Amalgam as a source of human exposure to mercury has been discussed in detail elsewhere (WHO, 1991) and will not be considered further in this document.

Disposition in the Body

The absorption of inorganic mercury from the diet was estimated by a WHO Expert Group (WHO, 1976) to be about 7% on the average. Most of the data came from Rahola et al., (1973) based on studies of 10 subjects. Individual differences were substantial, with absorption rates ranging perhaps as high as 25% in one subject. Animal studies indicated that inorganic mercury may be absorbed to a greater extent in the suckling period (Kostial et al., 1983).

The percentage of the absorbed dose that is deposited in the blood compartment, less than 0.5%, is much less than is the case with methylmercury, 5.0%. The disposition of inorganic mercury in other tissues and organs also differs from that of methylmercury. Accumulation in red blood cells, into hair, brain and fetal tissues, is much less than for the organic species. Inorganic mercury does, however, accumulate in placental tissue. The kidney is the main site of deposition, with the highest concentrations found in the kidney cortex.

Inorganic mercury is excreted in urine and feces. Inorganic mercury in urine is due to release from kidney tissue. It does not enter urine directly by glomerular filtration of mercury in plasma. Mercury in feces comes in part from the unabsorbed fraction in the diet and from biliary secretion. Like methylmercury, it is secreted from liver cells into bile as the glutathione complex, a process that does not begin until after weaning. Thus, young infants may be at a higher risk of accumulating inorganic mercury than adults because of both greater absorption and lower excretion. The suckling infant may be of special concern as inorganic mercury is secreted in breast milk.

Urine and plasma are the biological media most commonly used to assess the body burden of inorganic mercury. Plasma is preferred to whole blood because methylmercury accumulates in the red cells and might confuse estimates for inorganic mercury. Plasma will represent recent exposures as the half-time is relatively short. Since urine indicates kidney levels, it gives a better picture of cumulative exposure as the half-time in this tissue is much longer than for plasma.

Unfortunately, data on biological half-times in humans is sparse so we have to use observations on people exposed to mercury vapour. The latter is rapidly converted in body tissues to inorganic mercury so that biological half-times measured in subjects who had inhaled mercury vapour may be applicable. On this basis, a WHO Expert Group (WHO, 1991) concluded that plasma mercury has two phases of elimination, one with a half-time of 2-4 days and the other of 15-30 days. Half-times in kidney (presumable also urine) and whole body are about 2 months.

Adverse Health Effects

Inorganic mercury in the form of mercuric chloride has been taken by mouth accidentally or suicidally. The lethal dose in human adults is about 1 g (Gleason et al., 1963). Mercuric chloride is a corrosive poison. It destroys the lining of the gastrointestinal tract. The victim experiences cardiovascular collapse, enters a stage of shock resulting in acute renal failure, which is the proximate cause of death.

Less information is available on chronic exposure to inorganic mercury compounds in the diet. We have to rely mainly on animal studies (ATSDR, 1998). High daily doses of inorganic mercury, usually in the form of mercuric chloride added to drinking water, can produce damage to many organs and tissues. Overall, the kidney is the principal target tissue and usually the only tissue affected at low doses. This was confirmed by a comprehensive study conducted by the National Toxicology Program of the National Institute of Environmental Health Sciences (NTP, 1993).

The use of skin-whitening creams containing inorganic mercury can result in kidney damage. There may be an immunological component as the kidney glomerular tissue is attacked by antibodies, causing leakage of plasma protein into the urine resulting in the nephritic syndrome. The anti-glomerular antibodies appear to be generated as part of an autoimmune response to inorganic mercury. Similar effects have been reproduced in certain susceptible strains of rats.

Acrodynia or Pinks Disease was commonly reported in children up to the time when Warkany and Hubbard (1953) determined that inorganic mercury was the cause. Inorganic mercury in the form of calomel (mercurous chloride) was routinely added to teething powders and used as a cathartic in children. Mercuric chloride was used as an antiseptic in diaper washes. As a result of exposure both by mouth and via the skin, children developed a syndrome characterized by pink cheeks, severe joint pains and photophobia.

It was usually detected when the distraught mother brought the child to medical attention because of incessant crying and sleepless nights. Of 1000 or so infants exposed to inorganic mercury, only one might develop the full blown syndrome of acrodynia. In susceptible children, the disease might occur at urine levels as low as 20 μg Hg/L. An important corollary is that one case of acrodynia might indicate a much larger number of exposed infants (Gotelli et al., 1985).

Following Warkany and Hubbard's seminal paper, the disease has virtually disappeared. Consequently, little is known about the underlying toxic mechanisms.

Risks of Adverse Health Effects

The ATSDR (1998) from examination of chronic toxicity tests on rats conducted by the National Toxicology Program (NTP, 1993) found that the highest daily dose having no toxic effect was 0.23 mg Hg/kg. On a body weight basis, this is equivalent to 0.23 x 1000 x 70 = 16100 μg Hg for a 70-kg adult human. The ATSDR applied the customary safety factor of 100 to arrive at a "minimal risk level" of 2 μg Hg/kg/day equivalent to a daily intake of 140 μg Hg for a 70-kg adult. This is far in excess of the average daily intake of inorganic mercury for the general population estimated as 4.2 μg Hg/day (WHO, 1991), and as 2.6 μg Hg/day in Canada (Table 1).

Consumers of marine mammals could experience high intakes of inorganic mercury possibly exceeding the ATSDR minimum risk level. However, inorganic mercury in seal liver is believed to be inert, and is probably non-absorbable. On the other hand, the form of inorganic mercury in whale meat is not known. Pilot whales have levels in meat reported to average about 1.5 ppm (Juhlshamn et al., 1987). A single whale dinner of 200 g would represent a dietary intake of 300 μg Hg. The daily average would depend upon how frequently whale dinners are consumed. Episodic consumption might result in high "peak" intakes.

Another important gap in our knowledge of risks from inorganic mercury in the diet is the potential accumulation in suckling infants, especially if their mothers consume marine mammal meat. Inorganic mercury secreted in breast milk may be well absorbed by the infant and not excreted.

APPROACHES AND CONCLUSIONS BY REGULATORY AGENCIES AND ORGANIZATIONS

Ever since public health concern was aroused by the finding of methylmercury in fish in the late 1960s, numerous expert committees have evaluated the potential health risks and promulgated advisories and regulations. A brief summary is given in the following section.

The Swedish Expert Group

This landmark publication (Swedish Expert Group, 1971) pioneered the conceptual approach to evaluating human health risks from methylmercury in fish, an approach that with minor variations has been followed ever since. Basically, the group combined dose-response relationship with quantitative relationships between exposure (e.g. daily intake) and dose (body burden of methylmercury). The dose-response relationship came mainly from human studies, but were supported by animal data where possible. The exposure dose relationship was also derived from human data.

At the time of their analysis, most of the human data on dose response came from the second Japanese outbreak in Niigata (Tsubaki and Irukayama, 1977). The group identified the cases with the lowest blood or hair levels that existed at the time of appearance of signs and symptoms of poisoning. It noted that prior levels may have been higher in view of the latency period between exposure and onset of observable clinical effects. The lowest blood and hair levels were identified as 200 μg Hg/L and 50 ppm, respectively, for non-pregnant adults.

Data from methylmercury metabolic studies on humans, mainly conducted in Sweden, were used to estimate that a blood level of 200 μg Hg/L would be attained by a long-term daily intake of about 300 μg Hg as methylmercury.

World Health Organization

In its first evaluation (WHO, 1972), the WHO basically followed the Swedish approach and came to the same conclusions. It applied a safety factor of 10 to the Swedish lowest effect levels to arrive at a "tolerable" weekly intake of 300 μg Hg, of which no more than 200 μg Hg should be in the form of methylmercury. This corresponds to approximately 30 μg Hg/day as methylmercury and would yield corresponding blood and hair levels of 20 μg Hg/L and 5 ppm, respectively, after long-term (about one year or more) daily exposure.

Since that time, two criteria documents have been written (WHO, 1976, 1990). The first document now had additional data on adult dose-response data from the Iraqi outbreak. It also had additional data on exposure-dose relationships. As these data confirmed the original figures used by the Swedish Expert Group, the first criteria document confirmed the lowest effect levels for adults as 200 μg Hg/L in blood and 50 ppm in hair.

The second criteria document had additional information on studies of adult exposures and, for the first time, dose-response relationships for prenatal exposures. Once more, the adult data confirmed the previous analyses, except perhaps that the adult no effect level might be too conservative. However, no changes were made to the established figures.

The dose-response data from prenatal exposures were now available from the Iraqi study, along with the results of the studies in Canada by the McGill group and the studies in New Zealand by the Kjellström group. Their estimates were based on the Iraqi reports, leading to the conclusion that "a prudent interpretation of the Iraqi data implies that a 5% risk may be associated with a peak maternal mercury level of 10-20 μg Hg in the maternal hair." They emphasized the need for further epidemiological studies in view of the statistical uncertainties in estimates based on the Iraqi data.

Food Directorate, Health Protection Branch (HPB) and the US Food and Drug Administration

These agencies are charged with assuring the safety and wholesomeness of food in the commercial market. In the case of toxic food additives, this is achieved by specifying maximum allowable concentrations in food items.

In Canada, the Food Directorate of HPB uses a level of 0.5 ppm which has been adopted by most provinces. In the United States, a limit of 0.5 ppm total mercury was also set for edible tissues

of fish, but later changed to 1 ppm mercury as methylmercury after a court challenge to the original level. The 1 ppm level has been in effect since 1978.

The derivation of such "guidelines" or "action" levels for mercury in fish follows complex calculations of methylmercury intake from fish. Surveys of dietary intake of various species of fish in the open market are combined with data on methylmercury levels in fish tissue. These data become the basis for estimates of methylmercury intake from fish in the general population. An upper concentration is then chosen that will restrict the daily intake of methylmercury to protect the majority of the population. However, these agencies must also take into account the beneficial nutritional aspects of fish so that risk-benefit consideration will determine the final number. In April 1998, Food Directorate HPB after reviewing the Seychelles and Faroe Island data set a temporary Tolerable Daily Intake (TDI) for women of child-bearing age and young children at 0.2 μg Hg/day/kg body weight. This temporary TDI will be revised after the Seychelles study has been completed.

US Environmental Protection Agency

This agency has recently completed a "Mercury Study Report to Congress" (U.S. EPA Dec. 1997) that estimates a "reference" dose for dietary intake of methylmercury. This reference dose is designed to be protective under virtually all circumstances for all subgroups of the U.S. population. It chose the Iraqi studies of prenatal exposures as the basis of its estimate. First, it calculated a "benchmark" hair level (roughly equivalent to a threshold level) as 11 ppm as the peak value in maternal hair during pregnancy. It applied a safety factor and converted the hair level to an equivalent long-term daily intake to arrive at a reference dose of 0.1 μg Hg/day/kg body weight. This approximates to a roughly four-fold reduction in advisory levels as compared to the original WHO recommendations.

Agency for Toxic Substances and Disease Registry (ATSDR)

This agency, a branch of the U.S. Public Health Service, publishes toxicological profiles of numerous toxic chemicals. Included in the profile is an estimate of the Minimal Risk Levels (MRL) expressed as a daily intake per kg body weight. They are essentially similar to the US EPA's reference dose, though perhaps not as extreme in terms of total removal of risks.

The ATSDR selected data from the Seychelles study as the basis for the MRL. It concluded

that the median maternal hair level of 5.9 ppm in the Seychelles was a no effect level for prenatal exposures. This is converted to an equivalent long-term daily intake of 0.5 μg Hg/day/kg body weight. This is essentially the same as the long-standing WHO tolerable weekly intake of 200 μg Hg/ day per adult.

It argued that the application of a safety factor was unnecessary as the prenatal studies were conducted on the most sensitive subgroup of the population.

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APPENDIX I

TABLES
MEDICAL SERVICES BRANCH

Table 1. Cumulative results - January 1, 1983 to December 31, 1996 - by region and level. 1
Table 1A. Cumulative results to December 31, 1996 - by region and level. 2
Table 1B. Cumulative individual results to December 31, 1996 - by region and level. 3
Table 2. Cumulative results to December 31, 1996 - by region, community and level. 4
Table 3. Results - January 1, 1983 to December 31, 1996
- by region, community, year and level. 25
Table 4. Total individuals > 100 ppb - January 1, 1983 to December 31, 1996
- by region and level. 50
Table 4A. Total individuals > 100 ppb to December 31, 1996
- by region and level. 51
Table 5. Total individuals > 100 ppb - January 1, 1983 to December 31, 1996
- by region, community and level. 52
Table 5A. Total individuals > 100 ppb to December 31, 1996
- by region, community and level. 53
Table 6. Total individuals > 100 ppb to December 31, 1996
- by region and year 56
Table 7. Total individuals > 100 ppb - January 1, 1983 to December 31, 1996
- by region, community, year and level. 58
Table 8. Total individuals > 100 ppb - January 1, 1983 to December 31, 1996
- by region, sex and age. 65
Table 8A. Total individuals > 100 ppb to December 31, 1996
- by region, sex and age. 66
Table 9. Total individuals > 100 ppb - January 1, 1983 to December 31, 1996
- by region, community, sex, age and level. 67
Table 9A. Total individuals > 100 ppb to December 31, 1996
- by region, community, sex, age and level 68
Table 10. Mother/child cord blood sampling results - January 1, 1983 to December 31, 1996
- by region and level. 71
Table 10A. Mother/child cord blood sampling results to December 31, 1996
- by region and level. 72

TABLE 1
TABLEAU 1

CUMULATIVE RESULTS JANUARY 1,1983 TO DECEMBER 31,1996 - BY REGION AND LEVEL
RÉSULTATS CUMULATIFS DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996 - PAR RÉGION ET NIVEAU

()Percentage of Total Tests
()Pourcentage du total des analyses

Region Région	No. of Communities Nbre de collectivités	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L	Highest Result Résultat le plus élevé	Year Année
ATLANTIC ATLANTIQUE	0	0	-	-	-	-	-	-	-	-			
QUEBEC QUÉBEC	25	2278	2083 (91.44)	195 (8.56)	-	-	-	-	-	-		99	1986
ONTARIO	41	4189	3424 (81.74)	738 (17.62)	27 (0.64)	-	-	-	-	-		173	1984
MANITOBA	44	6410	5543 (86.47)	855 (13.34)	11 (0.17)	1 (0.02)	-	-	-	-		262	1989
SASKATCHEWAN	3	56	55 (98.21)	1 (1.79)	-	-	-	-	-	-		29	1983
ALBERTA	2	167	167 (100.00)	-	-	-	-	-	-	-		18	1989
BRITISH COLUMBIA COLOMBIE BRITANNIQUE	11	365	351 (96.16)	14 (3.84)	-	-	-	-	-	-		60	1991
NORTHWEST TERRITORIES TERRITOIRES DU NORD-OUEST	10	411	200 (48.66)	204 (49.64)	6 (1.46)	1 (0.24)	-	-	-	-		218	1984
YUKON	0	0	-	-	-	-	-	-	-	-		-	
Total	136	13876	11823 (85.21)	2007 (14.46)	44 (0.32)	2 (0.01)	-	-	-	-			

TABLE 1A
TABLEAU 1A

CUMULATIVE RESULTS TO DECEMBER 31,1996 - BY REGION AND LEVEL
RÉSULTATS CUMULATIFS AU 31 DÉCEMBRE 1996 - PAR RÉGION ET NIVEAU

()Percentage of Total Tests
()Pourcentage du total des analyses

Region Région	No. of Communities Nbre de collectivités	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L	Highest Result Résultat le plus élevé	Year Année
ATLANTIC ATLANTIQUE	23	710	695 (97.89)	15 (2.11)	- -	- -	- -	- -	- -	- -		99	1978
QUEBEC QUÉBEC	52	23638	14570 (61.64)	8379 (35.45)	609 (2.58)	57 (0.24)	13 (0.05)	5 (0.02)	4 (0.02)	1 (0.00)		649	1975
ONTARIO	106	20867	16802 (80.52)	3779 (18.11)	239 (1.15)	32 (0.15)	8 (0.04)	5 (0.02)	- -	2 (0.01)		660	1971
MANITOBA	69	13902	11569 (83.22)	2297 (16.52)	34 (0.24)	2 (0.01)	- -	- -	- -	- -		262	1989
SASKATCHEWAN	74	2505	2248 (89.74)	252 (10.06)	5 (0.20)	- -	- -	- -	- -	- -		124	1978
ALBERTA	38	1505	1451 (96.41)	52 (3.46)	2 (0.13)	- -	- -	- -	- -	- -		105	1977
BRITISH COLUMBIA COLOMBIE BRITANNIQUE	90	4740	4422 (93.29)	315 (6.65)	3 (0.06)	- -	- -	- -	- -	- -		146	1978
NORTHWEST TERRITORIES TERRITOIRES DU NORD-OUEST	59	3827	2273 (59.39)	1515 (39.59)	33 (0.86)	3 (0.08)	3 (0.08)	- -	- -	- -		363	1971
YUKON	18	862	855 (99.19)	7 (0.81)	- -	- -	- -	- -	- -	- -		67	1977
Total	529	72556	54885 (75.65)	16611 (22.89)	925 (1.28)	94 (0.13)	24 (0.03)	10 (0.01)	4 (0.01)	3 (0.00)			

TABLE 1B
TABLEAU 1B

CUMULATIVE INDIVIDUAL RESULTS TO DECEMBER 31,1996 - BY REGION AND LEVEL
RÉSULTATS CUMULATIFS INDIVIDUELS AU 31 DÉCEMBRE 1996 - PAR RÉGION ET NIVEAU

()Percentage of Total Tests
()Pourcentage du total des analyses

Region Région	Number of individuals nombre de personne	0-19	20-100	> 100	ppb µg/L
ATLANTIC ATLANTIQUE	700	683 (97.57)	17 (2.43)	-	
QUEBEC QUÉBEC	9514	5406 (56.82)	3686 (38.74)	422 (4.44)	
ONTARIO	12431	10146 (81.62)	2154 (17.33)	131 (1.05)	
MANITOBA	9835	8566 (87.10)	1250 (12.71)	19 (0.19)	
SASKATCHEWAN	1790	1578 (88.16)	207 (11.56)	5 (0.28)	
ALBERTA	801	772 (96.38)	28 (3.50)	1 (0.12)	
BRITISH COLUMBIA COLOMBIE BRITANNIQUE	2970	2737 (92.16)	230 (7.74)	3 (0.10)	
NORTHWEST TERRITORIES TERRITOIRES DU NORD-OUEST	2294	1251 (54.53)	1009 (43.99)	34 (1.48)	
YUKON	299	291 (97.32)	8 (2.68)	-	
Total	40634	31430 (77.35)	8589 (21.14)	615 (1.51)	

TABLE 2
TABLEAU 2

CUMULATIVE RESULTS TO DECEMBER 31, 1996 - BY REGION, COMMUNITY AND LEVEL
RÉSULTATS CUMULATIFS AU 31 DÉCEMBRE 1996 - PAR RÉGION, COLLECTIVITÉ ET NIVEAU

ATLANTIC - ATLANTIQUE Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Big Cove	132	126	6	-	-	-	-	-	-	
Buctouche	7	7	-	-	-	-	-	-	-	
Burnt Church	74	74	-	-	-	-	-	-	-	
Eel Ground	56	56	-	-	-	-	-	-	-	
Eel River Bar	84	84	-	-	-	-	-	-	-	
Eskasoni	40	40	-	-	-	-	-	-	-	
Fort Folly	10	7	3	-	-	-	-	-	-	
Indian Island	14	14	-	-	-	-	-	-	-	
Kingsclear	39	36	3	-	-	-	-	-	-	
Lennox Island	23	23	-	-	-	-	-	-	-	
Morell	6	6	-	-	-	-	-	-	-	
Oromocto	17	17	-	-	-	-	-	-	-	
Pabineau	20	20	-	-	-	-	-	-	-	
Pictou Landing	4	4	-	-	-	-	-	-	-	
Red Bank	38	38	-	-	-	-	-	-	-	
Rocky Point	2	1	1	-	-	-	-	-	-	
Scotch Fort	9	8	1	-	-	-	-	-	-	
St Basile	12	12	-	-	-	-	-	-	-	
St Mary's	36	36	-	-	-	-	-	-	-	
Tobique	57	57	-	-	-	-	-	-	-	
Wagmatcook	3	3	-	-	-	-	-	-	-	
Whycocomagh	9	8	1	-	-	-	-	-	-	
Woodstock	18	18	-	-	-	-	-	-	-	
Total	710	695	15	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

QUEBEC - QUÉBEC Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Abitibi Dominium (Pikogan)	228	166	51	6	3	2	-	-	-	
Akulivik	28	2	26	-	-	-	-	-	-	
Aupaluk	16	5	11	-	-	-	-	-	-	
Bécancour (Wolinak)	70	70	-	-	-	-	-	-	-	
Bersimis (Betsiamites)	325	321	4	-	-	-	-	-	-	
Caughnawaga (Kahnawake)	173	172	1	-	-	-	-	-	-	
Chapais (Matagami/Chapais)	12	5	7	-	-	-	-	-	-	
Chibougamau	35	12	23	-	-	-	-	-	-	
Eastmain	356	269	87	-	-	-	-	-	-	
Escoumains (Les Escoumins)	92	87	5	-	-	-	-	-	-	
Fort George	2201	1515	637	40	8	1	-	-	-	
Inukjuaq (UnukJuaq)	694	133	539	22	-	-	-	-	-	
Ivujivik	55	5	42	8	-	-	-	-	-	
Kangirsujuak (Wakeham Bay)	184	68	114	2	-	-	-	-	-	
Kangirsuk	128	45	83	-	-	-	-	-	-	
Kipawa	175	171	4	-	-	-	-	-	-	
Kujuarapik (Great Whales)	994	456	458	68	11	-	1	-	-	
Kuujjuaq (Fort Chimo)	323	221	101	1	-	-	-	-	-	
Lac Simon	300	202	90	7	1	-	-	-	-	
Lorette (Village Huron)	138	136	2	-	-	-	-	-	-	
Maliotenam	117	113	4	-	-	-	-	-	-	
Maniwaki (River Desert)	103	101	1	1	-	-	-	-	-	
Manouan	788	564	220	4	-	-	-	-	-	
Maria	208	203	5	-	-	-	-	-	-	
Matagami (Matagami/Chapais)	8	3	5	-	-	-	-	-	-	
Mingan	169	161	8	-	-	-	-	-	-	
Miquelon	12	7	5	-	-	-	-	-	-	
Mistassini	6612	3719	2640	237	15	1	-	-	-	
Natashquan	134	129	5	-	-	-	-	-	-	
Notre-Dame-du-Nord(Timiskaming)	220	217	3	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

QUEBEC - QUÉBEC Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Obedjinan	1162	639	508	13	2	-	-	-	-	
Odanak	100	99	1	-	-	-	-	-	-	
Paint Hills (Wemindji)	626	370	250	4	1	1	-	-	-	
Pointe Bleue	468	421	47	-	-	-	-	-	-	
Port-Nouveau Québec	140	103	37	-	-	-	-	-	-	
Puvirnituq (Povungnituk)	415	152	262	1	-	-	-	-	-	
Quartaq (Koartak)	123	18	97	8	-	-	-	-	-	
Rapide-des-Cèdres	11	2	9	-	-	-	-	-	-	
Rapid Lake	140	125	15	-	-	-	-	-	-	
Restigouche	147	143	4	-	-	-	-	-	-	
Romaine	217	197	20	-	-	-	-	-	-	
Rupert House (Waskagonish)	682	511	165	6	-	-	-	-	-	
St. Augustin	109	94	15	-	-	-	-	-	-	
Salluit (Sugluk)	842	199	562	75	6	-	-	-	-	
Schefferville	620	304	295	20	1	-	-	-	-	
Senneterre	4	4	-	-	-	-	-	-	-	
Sept Iles	119	112	7	-	-	-	-	-	-	
Tasuijaq (Leaf Bay)	92	38	54	-	-	-	-	-	-	
Waswanipi	2181	1295	774	86	9	8	4	4	1	
Weymontachie	203	188	15	-	-	-	-	-	-	
Winneway (Longue-Pointe)	318	259	59	-	-	-	-	-	-	
Wolf Lake	21	19	2	-	-	-	-	-	-	
Total	23638	14570	8379	609	57	13	5	4	1	

TABLE 2
TABLEAU 2

ONTARIO Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Alderville	8	8	-	-	-	-	-	-	-	
Angle Inlet	2	2	-	-	-	-	-	-	-	
Angling Lake	57	43	14	-	-	-	-	-	-	
Attawapiskat	359	329	29	1	-	-	-	-	-	
Batchewana (Rankin Reserve)	9	7	2	-	-	-	-	-	-	
Bear Island	2	2	-	-	-	-	-	-	-	
Bearskin Lake	145	103	42	-	-	-	-	-	-	
Big Grassy River	43	42	1	-	-	-	-	-	-	
Big Island	29	29	-	-	-	-	-	-	-	
Big Trout Lake	484	299	185	-	-	-	-	-	-	
Birch Island (Whitefish River)	122	122	-	-	-	-	-	-	-	
Cape Croker	2	2	-	-	-	-	-	-	-	
Cat Lake	134	108	26	-	-	-	-	-	-	
Chapleau	31	31	-	-	-	-	-	-	-	
Collins	10	8	2	-	-	-	-	-	-	
Constance Lake	98	95	3	-	-	-	-	-	-	
Couchiching	177	173	4	-	-	-	-	-	-	
Curve Lake	2	2	-	-	-	-	-	-	-	
Dalles	2	1	1	-	-	-	-	-	-	
Deer Lake	351	216	129	6	-	-	-	-	-	
Dokis	86	86	-	-	-	-	-	-	-	
Duck Lake	37	37	-	-	-	-	-	-	-	
Eagle Lake	27	25	1	1	-	-	-	-	-	
Fort Albany	298	290	8	-	-	-	-	-	-	
Fort Hope	518	352	166	-	-	-	-	-	-	
Fort Severn	151	147	4	-	-	-	-	-	-	
Fort William	26	26	-	-	-	-	-	-	-	
Garden River	152	151	1	-	-	-	-	-	-	
Georgina Island	10	10	-	-	-	-	-	-	-	
Gibson	32	32	-	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

ONTARIO Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Golden Lake	57	56	1	-	-	-	-	-	-	
Grassy Narrows	4050	2871	987	154	26	6	5	-	1	
Gull Bay	59	53	6	-	-	-	-	-	-	
Hawley Lake	7	7	-	-	-	-	-	-	-	
Henvey Inlet - Bakanon	24	24	-	-	-	-	-	-	-	
- Pickeral	30	29	1	-	-	-	-	-	-	
Hiawatha	10	10	-	-	-	-	-	-	-	
Hudson	10	10	-	-	-	-	-	-	-	
Kasabonika	282	134	148	-	-	-	-	-	-	
Kashechewan	290	285	5	-	-	-	-	-	-	
Kettle Point	15	14	1	-	-	-	-	-	-	
Kingfisher Lake	127	101	26	-	-	-	-	-	-	
Lac La Croix	138	118	20	-	-	-	-	-	-	
Lac Seul (Frenchmans Head)	314	242	72	-	-	-	-	-	-	
Lake Helen	30	28	2	-	-	-	-	-	-	
Lansdowne House	96	58	38	-	-	-	-	-	-	
Long Lake 58	224	219	5	-	-	-	-	-	-	
Long Lake 77	56	56	-	-	-	-	-	-	-	
Magnetawan	26	26	-	-	-	-	-	-	-	
Manitou Rapids	42	42	-	-	-	-	-	-	-	
Mattagami	54	44	10	-	-	-	-	-	-	
McDiarmid	1	1	-	-	-	-	-	-	-	
McDowell Lake	18	14	4	-	-	-	-	-	-	
Mckenzie Portage	15	14	1	-	-	-	-	-	-	
Michipicoten	17	17	-	-	-	-	-	-	-	
Mississauga	74	73	1	-	-	-	-	-	-	
Mobert (Pic Mobert)	84	80	4	-	-	-	-	-	-	
Moose Deer Point	32	31	1	-	-	-	-	-	-	
Muskrat Dam	105	86	19	-	-	-	-	-	-	
New Osnaburgh	312	278	34	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

ONTARIO Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Nipissing	65	64	1	-	-	-	-	-	-	
North Spirit Lake	128	75	51	2	-	-	-	-	-	
Northwest Angle 33	93	85	8	-	-	-	-	-	-	
Northwest Bay	39	39	-	-	-	-	-	-	-	
Ogoki Post	286	153	132	1	-	-	-	-	-	
Parry Island	24	21	3	-	-	-	-	-	-	
Pays Plat	15	13	2	-	-	-	-	-	-	
Pic Heron (Heron Bay Reserve)	154	143	11	-	-	-	-	-	-	
Pickle Lake	1	1	-	-	-	-	-	-	-	
Pikangikum	286	243	43	-	-	-	-	-	-	
Poplar Hill	102	48	47	6	1	-	-	-	-	
Rama (Chippewas of Rama)	9	9	-	-	-	-	-	-	-	
Rat Portage	183	178	5	-	-	-	-	-	-	
Red Gut Bay	58	54	4	-	-	-	-	-	-	
Round Lake (Weagamow Lake)	343	251	92	-	-	-	-	-	-	
Sabaskong	90	90	-	-	-	-	-	-	-	
Sachigo Lake	106	82	26	-	-	-	-	-	-	
Sandy Lake - Ontario	808	679	129	-	-	-	-	-	-	
Sarnia	16	16	-	-	-	-	-	-	-	
Saugeen (Chippewa Hill)	66	66	-	-	-	-	-	-	-	
Savant Lake	11	11	-	-	-	-	-	-	-	
Seine River	148	121	27	-	-	-	-	-	-	
Serpent River	111	110	1	-	-	-	-	-	-	
Shawanaga	27	27	-	-	-	-	-	-	-	
Sheguiandah	24	24	-	-	-	-	-	-	-	
Sheshegwaning	55	55	-	-	-	-	-	-	-	
Shoal Lake	174	174	-	-	-	-	-	-	-	
Six Nations Reserve	13	13	-	-	-	-	-	-	-	
Slate Falls	53	33	20	-	-	-	-	-	-	
Spanish River	211	211	-	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

ONTARIO Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Sucker Creek	36	35	1	-	-	-	-	-	-	
Summer Beaver	119	85	34	-	-	-	-	-	-	
Thessalon	20	20	-	-	-	-	-	-	-	
Tyendinaga	15	15	-	-	-	-	-	-	-	
Wabigoon Lake	24	17	7	-	-	-	-	-	-	
Walpole Island	281	252	29	-	-	-	-	-	-	
Washagamis Bay	16	16	-	-	-	-	-	-	-	
Webequie	323	256	67	-	-	-	-	-	-	
West Bay	113	113	-	-	-	-	-	-	-	
Whitedog	5017	3970	971	68	5	2	-	-	1	
Whitefish Bay	420	415	5	-	-	-	-	-	-	
Whitefish Lake	50	50	-	-	-	-	-	-	-	
Whitesands River (Armstrong)	7	7	-	-	-	-	-	-	-	
Wikwemikong	437	434	3	-	-	-	-	-	-	
Winisk (Peawanuck)	251	207	44	-	-	-	-	-	-	
Wunnumin	34	22	12	-	-	-	-	-	-	
Total	17463	14024	3161	231	32	8	5	-	2	

TABLE 2
TABLEAU 2

MANITOBA Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Anama Bay	45	43	2	-	-	-	-	-	-	
Berens River	116	114	2	-	-	-	-	-	-	
Birdtail Sioux	30	30	-	-	-	-	-	-	-	
Bloodvein	137	117	20	-	-	-	-	-	-	
Brochet	59	54	5	-	-	-	-	-	-	
Brokenhead	2	2	-	-	-	-	-	-	-	
Chemahawin (Easterville)	111	89	22	-	-	-	-	-	-	
Churchill	5	5	-	-	-	-	-	-	-	
Crane River	36	36	-	-	-	-	-	-	-	
Cross Lake	1572	1460	110	1	1	-	-	-	-	
Dakota Plains	41	39	1	-	1	-	-	-	-	
Dakota Tipi	16	15	1	-	-	-	-	-	-	
Dauphin River	24	23	1	-	-	-	-	-	-	
Ebb and Flow	55	55	-	-	-	-	-	-	-	
Fairford	101	101	-	-	-	-	-	-	-	
Fisher River	102	102	-	-	-	-	-	-	-	
Fort Alexander	214	209	5	-	-	-	-	-	-	
Fox Lake (Manitoba)	67	45	20	2	-	-	-	-	-	
Garden Hill	185	155	30	-	-	-	-	-	-	
God's Lake	301	194	107	-	-	-	-	-	-	
God's River	201	63	138	-	-	-	-	-	-	
Grand Rapids	30	27	3	-	-	-	-	-	-	
Granville Lake	82	37	45	-	-	-	-	-	-	
Gypsumville	1	1	-	-	-	-	-	-	-	
Hollow Water	156	147	9	-	-	-	-	-	-	
Ilford	10	9	1	-	-	-	-	-	-	
Jackhead	53	52	1	-	-	-	-	-	-	
Keeseekoowenin	34	31	3	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

MANITOBA Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Lac Brochet	331	174	156	1	-	-	-	-	-	
Lac du Bonnet	1	1	-	-	-	-	-	-	-	
Lake Manitoba	49	49	-	-	-	-	-	-	-	
Lake St. Martin	82	80	2	-	-	-	-	-	-	
Little Black River	88	87	1	-	-	-	-	-	-	
Little Grand Rapids	210	145	65	-	-	-	-	-	-	
Little Saskatchewan	36	34	2	-	-	-	-	-	-	
Long Plain	63	63	-	-	-	-	-	-	-	
Moose Lake	107	98	9	-	-	-	-	-	-	
Nelson House	1755	1416	336	3	-	-	-	-	-	
Norway House	1664	1581	83	-	-	-	-	-	-	
Oak Lake	48	47	1	-	-	-	-	-	-	
Oxford House	65	56	9	-	-	-	-	-	-	
Pauingassi	216	105	94	17	-	-	-	-	-	
Peguis	125	120	5	-	-	-	-	-	-	
Pine Creek	127	123	4	-	-	-	-	-	-	
Pointe Du Bois	12	5	7	-	-	-	-	-	-	
Poplar River	70	70	-	-	-	-	-	-	-	
Princess Harbour	2	2	-	-	-	-	-	-	-	
Pukatawagan	314	186	128	-	-	-	-	-	-	
Red Sucker Lake	140	90	50	-	-	-	-	-	-	
Rolling River	40	40	-	-	-	-	-	-	-	
Roseau River	57	56	1	-	-	-	-	-	-	
St. Theresa Point	329	271	58	-	-	-	-	-	-	
Sandy Bay	68	68	-	-	-	-	-	-	-	
Seymourville	1	1	-	-	-	-	-	-	-	
Shamattawa	204	163	41	-	-	-	-	-	-	
Shoal River	125	124	1	-	-	-	-	-	-	
Sioux Valley	85	84	1	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

MANITOBA Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
South Indian Lake	1252	843	403	6	-	-	-	-	-	
Split Lake	1170	1018	152	-	-	-	-	-	-	
Swan Lake	45	45	-	-	-	-	-	-	-	
Sypsumville	25	25	-	-	-	-	-	-	-	
Tadoule Lake	103	64	39	-	-	-	-	-	-	
The Pas	218	210	8	-	-	-	-	-	-	
Valley River	79	75	4	-	-	-	-	-	-	
Wasagamack	132	105	27	-	-	-	-	-	-	
Waterhen	44	44	-	-	-	-	-	-	-	
Waywayseecappo	90	88	2	-	-	-	-	-	-	
York Landing	544	458	82	4	-	-	-	-	-	
Total	13902	11569	2297	34	2	-	-	-	-	

TABLE 2
TABLEAU 2

SASKATCHEWAN Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Assiniboine (Carry The Kettle)	9	9	-	-	-	-	-	-	-	
Ahtahkakoop	39	39	-	-	-	-	-	-	-	
Beardy's And Okemasis	43	43	-	-	-	-	-	-	-	
Big Head (Joseph Bighead)	12	12	-	-	-	-	-	-	-	
Big River	40	40	-	-	-	-	-	-	-	
Buffalo River	31	31	-	-	-	-	-	-	-	
Canoe Lake	57	57	-	-	-	-	-	-	-	
Chicken (Black Lake)	53	38	15	-	-	-	-	-	-	
Chitek Lake (Pelican Lake)	104	103	1	-	-	-	-	-	-	
Cote	11	11	-	-	-	-	-	-	-	
Cowessess	14	14	-	-	-	-	-	-	-	
Cumberland House	144	139	5	-	-	-	-	-	-	
Day Star	3	3	-	-	-	-	-	-	-	
Fishing Lake	4	4	-	-	-	-	-	-	-	
Flying Dust	15	15	-	-	-	-	-	-	-	
Fond du Lac	33	33	-	-	-	-	-	-	-	
Gordon	13	13	-	-	-	-	-	-	-	
Grandmother's Bay	12	5	7	-	-	-	-	-	-	
James Smith	39	39	-	-	-	-	-	-	-	
Kahkewistahaw	17	14	2	1	-	-	-	-	-	
Keeseekoose	14	14	-	-	-	-	-	-	-	
Kinistino	3	3	-	-	-	-	-	-	-	
Kinoosao	13	6	6	1	-	-	-	-	-	
Lac La Hache	38	10	28	-	-	-	-	-	-	
Lac La Ronge	85	68	16	1	-	-	-	-	-	
Little Black Bear	8	7	-	1	-	-	-	-	-	
Little Bone	1	1	-	-	-	-	-	-	-	
Little Pine & Luckyman	32	32	-	-	-	-	-	-	-	
Little Red River	24	24	-	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

SASKATCHEWAN Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Makwa Lake	19	19	-	-	-	-	-	-	-	
Ministikwan	6	6	-	-	-	-	-	-	-	
Mistawasis	34	32	1	1	-	-	-	-	-	
Montreal Lake	29	28	1	-	-	-	-	-	-	
Mosquito	28	28	-	-	-	-	-	-	-	
Muscowpetung	19	19	-	-	-	-	-	-	-	
Muskeg Lake	17	17	-	-	-	-	-	-	-	
Muskoday	19	19	-	-	-	-	-	-	-	
Muskowekwan	8	8	-	-	-	-	-	-	-	
New Moosomin (Moosomin)	31	30	1	-	-	-	-	-	-	
New Thunderchild	28	27	1	-	-	-	-	-	-	
Nikaneet	6	6	-	-	-	-	-	-	-	
Nut Lake	8	7	1	-	-	-	-	-	-	
Ochapowace	12	12	-	-	-	-	-	-	-	
Okanese	5	5	-	-	-	-	-	-	-	
One Arrow	18	18	-	-	-	-	-	-	-	
Pasqua	31	29	2	-	-	-	-	-	-	
Peepeekisis	17	17	-	-	-	-	-	-	-	
Pelican Narrows	288	254	34	-	-	-	-	-	-	
Piapot	35	35	-	-	-	-	-	-	-	
Poorman	11	11	-	-	-	-	-	-	-	
Portage La Loche (La Loche)	20	20	-	-	-	-	-	-	-	
Poundmaker	24	24	-	-	-	-	-	-	-	
Red Earth	79	79	-	-	-	-	-	-	-	
Red Pheasant	32	31	1	-	-	-	-	-	-	
Sakimay	5	5	-	-	-	-	-	-	-	
Sandy Bay	1	1	-	-	-	-	-	-	-	
Saulteaux	10	9	1	-	-	-	-	-	-	
Saulteaux (Birch Lake)	1	1	-	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

SASKATCHEWAN Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Seekaskootch	44	43	1	-	-	-	-	-	-	
Shoal Lake	160	160	-	-	-	-	-	-	-	
Southend	217	121	96	-	-	-	-	-	-	
Standing Buffalo	52	52	-	-	-	-	-	-	-	
Stanley	49	26	23	-	-	-	-	-	-	
Star Blanket	6	5	1	-	-	-	-	-	-	
Sturgeon Lake	30	30	-	-	-	-	-	-	-	
Sturgeon Weir	15	14	1	-	-	-	-	-	-	
Sweet Grass	1	1	-	-	-	-	-	-	-	
The Key	5	5	-	-	-	-	-	-	-	
Turnor Lake	19	17	2	-	-	-	-	-	-	
Wahpaton	13	13	-	-	-	-	-	-	-	
Wapachewunak	24	21	3	-	-	-	-	-	-	
Waterhen	78	77	1	-	-	-	-	-	-	
White Bear	26	25	1	-	-	-	-	-	-	
Witchekan Lake	14	14	-	-	-	-	-	-	-	
Total	2505	2248	252	5	-	-	-	-	-	

TABLE 2
TABLEAU 2

ALBERTA Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Alexander	14	14	-	-	-	-	-	-	-	
Alexis	10	10	-	-	-	-	-	-	-	
Beaver Lake	43	42	1	-	-	-	-	-	-	
Blackfoot	69	69	-	-	-	-	-	-	-	
Blood	146	143	1	2	-	-	-	-	-	
Chipewyan Lake	12	12	-	-	-	-	-	-	-	
Cols Lake	29	29	-	-	-	-	-	-	-	
Driftpile	20	20	-	-	-	-	-	-	-	
Enoch	9	9	-	-	-	-	-	-	-	
Ermineskin	5	5	-	-	-	-	-	-	-	
Fitzgerald	4	3	1	-	-	-	-	-	-	
Fort Chipewyan	97	87	10	-	-	-	-	-	-	
Fort Mckay	43	39	4	-	-	-	-	-	-	
Fox Lake	53	51	2	-	-	-	-	-	-	
Frog Lake	17	16	1	-	-	-	-	-	-	
Garden River	6	4	2	-	-	-	-	-	-	
Gleichan	1	1	-	-	-	-	-	-	-	
Goodfish Lake	86	86	-	-	-	-	-	-	-	
Hay Lake	112	98	14	-	-	-	-	-	-	
Janvier	20	15	5	-	-	-	-	-	-	
Jean D'or Prairie	17	16	1	-	-	-	-	-	-	
Kehewin	3	3	-	-	-	-	-	-	-	
Louis Bull	4	4	-	-	-	-	-	-	-	
Paul Band (Wabamun)	6	6	-	-	-	-	-	-	-	
Peerless Lake	12	10	2	-	-	-	-	-	-	
Peigan	211	210	1	-	-	-	-	-	-	
Saddle Lake	160	158	2	-	-	-	-	-	-	
Samson	19	19	-	-	-	-	-	-	-	
Sandy Lake	15	15	-	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

ALBERTA Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Sarcee	6	6	-	-	-	-	-	-	-	
Stoney	3	3	-	-	-	-	-	-	-	
Sturgeon Lake	97	97	-	-	-	-	-	-	-	
Sucker Creek	9	9	-	-	-	-	-	-	-	
Sunchild	15	15	-	-	-	-	-	-	-	
Swan River	5	5	-	-	-	-	-	-	-	
Trout Lake	8	8	-	-	-	-	-	-	-	
Wabasca/Desmarais	106	101	5	-	-	-	-	-	-	
Whitefish Lake	13	13	-	-	-	-	-	-	-	
Total	1505	1451	52	2	-	-	-	-	-	

TABLE 2
TABLEAU 2

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Ahousaht	20	20	-	-	-	-	-	-	-	
Anacla	18	18	-	-	-	-	-	-	-	
Anahim Lake	15	15	-	-	-	-	-	-	-	
Babine	62	60	2	-	-	-	-	-	-	
Blueberry	16	16	-	-	-	-	-	-	-	
Buckley House	6	1	5	-	-	-	-	-	-	
Burns Lake	2	2	-	-	-	-	-	-	-	
Campbell River	10	10	-	-	-	-	-	-	-	
Canoe Creek	10	10	-	-	-	-	-	-	-	
Canyon City	72	72	-	-	-	-	-	-	-	
Cape Mudge	11	11	-	-	-	-	-	-	-	
Chemainus	43	43	-	-	-	-	-	-	-	
Cheslatta	35	35	-	-	-	-	-	-	-	
Church House	7	7	-	-	-	-	-	-	-	
Comox	6	6	-	-	-	-	-	-	-	
Cowichan	217	210	7	-	-	-	-	-	-	
Creston	26	26	-	-	-	-	-	-	-	
Dog Creek	12	12	-	-	-	-	-	-	-	
Fort Rupert	16	15	1	-	-	-	-	-	-	
Fort Ware	179	169	10	-	-	-	-	-	-	
Fountain	10	10	-	-	-	-	-	-	-	
Fraser Lake	1	1	-	-	-	-	-	-	-	
Gilford Island	13	12	1	-	-	-	-	-	-	
Gold River	12	12	-	-	-	-	-	-	-	
Greenville	195	173	22	-	-	-	-	-	-	
Halalt	1	1	-	-	-	-	-	-	-	
Hazelton	22	22	-	-	-	-	-	-	-	
Ingenika	335	296	39	-	-	-	-	-	-	
Iskut	21	21	-	-	-	-	-	-	-	
Kincolith	277	220	57	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE

Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Kingcome Inlet	16	16	-	-	-	-	-	-	-	
Kitimaat	107	102	5	-	-	-	-	-	-	
Kitkatla	168	160	8	-	-	-	-	-	-	
Kitselas	6	6	-	-	-	-	-	-	-	
Kitsumkaylum	13	13	-	-	-	-	-	-	-	
Kyuquot	17	17	-	-	-	-	-	-	-	
Lillooet	6	6	-	-	-	-	-	-	-	
Malahat	8	7	1	-	-	-	-	-	-	
Masset	135	135	-	-	-	-	-	-	-	
McLeod Lake	25	25	-	-	-	-	-	-	-	
Metlakatla	14	14	-	-	-	-	-	-	-	
Moberly Lake East	6	6	-	-	-	-	-	-	-	
Moberly Lake West	9	9	-	-	-	-	-	-	-	
Mount Currie	22	22	-	-	-	-	-	-	-	
Mowachaht	3	3	-	-	-	-	-	-	-	
Nanaimo	54	53	1	-	-	-	-	-	-	
Nanoose	12	12	-	-	-	-	-	-	-	
Nautley	17	17	-	-	-	-	-	-	-	
Necoslie	356	328	27	1	-	-	-	-	-	
Nee-Tahi-Buhn	2	2	-	-	-	-	-	-	-	
New Aiyansh	303	299	4	-	-	-	-	-	-	
New Quatsino	16	16	-	-	-	-	-	-	-	
Nimpkish	128	119	8	1	-	-	-	-	-	
Nitinaht	8	8	-	-	-	-	-	-	-	
Nuchatlaht	3	3	-	-	-	-	-	-	-	
Ohiat	1	1	-	-	-	-	-	-	-	
Opetchesaht (Ahahswinis)	9	9	-	-	-	-	-	-	-	
Pacheenaht	12	11	1	-	-	-	-	-	-	
Pat Bay	1	1	-	-	-	-	-	-	-	
Pauquachin (Cole Bay)	17	16	1	-	-	-	-	-	-	
Pavilion	6	6	-	-	-	-	-	-	-	

TABLE 2
TABLEAU 2

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Penelakut	1	1	-	-	-	-	-	-	-	
Pinchi & Tachi	601	528	73	-	-	-	-	-	-	
Portage	89	80	9	-	-	-	-	-	-	
Qualicum	12	10	2	-	-	-	-	-	-	
Quanichan	1	1	-	-	-	-	-	-	-	
Queen's Cove	10	10	-	-	-	-	-	-	-	
River Road	11	11	-	-	-	-	-	-	-	
St Mary's	44	44	-	-	-	-	-	-	-	
Seaspunkat	6	6	-	-	-	-	-	-	-	
Sheshaht (Tsahaheh)	48	46	1	1	-	-	-	-	-	
Shuswap	1	1	-	-	-	-	-	-	-	
Skidegate	56	56	-	-	-	-	-	-	-	
Sliammon	21	21	-	-	-	-	-	-	-	
Sooke	3	3	-	-	-	-	-	-	-	
Squamish	50	50	-	-	-	-	-	-	-	
Squirrel Cove	5	5	-	-	-	-	-	-	-	
Stellaquo	1	1	-	-	-	-	-	-	-	
Stoney Creek	2	2	-	-	-	-	-	-	-	
Tachet	21	21	-	-	-	-	-	-	-	
Takla Landing	323	303	20	-	-	-	-	-	-	
Tobacco Plains	7	7	-	-	-	-	-	-	-	
Topley Landing	29	27	2	-	-	-	-	-	-	
Tsartlip (South Saanish)	45	44	1	-	-	-	-	-	-	
Tsawout (East Saanich)	28	28	-	-	-	-	-	-	-	
Tseycum (Union Bay)	6	6	-	-	-	-	-	-	-	
Tsulquate	44	38	6	-	-	-	-	-	-	
Ucluelet	20	20	-	-	-	-	-	-	-	
Westholme	14	13	1	-	-	-	-	-	-	
Woyenne	71	71	-	-	-	-	-	-	-	
Total	4740	4422	315	3	-	-	-	-	-	

TABLE 2
TABLEAU 2

NORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST

Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Aklavik	39	26	13	-	-	-	-	-	-	
Arctic Bay	406	200	204	2	-	-	-	-	-	
Arctic Red River	10	7	3	-	-	-	-	-	-	
Baker Lake	15	9	6	-	-	-	-	-	-	
Broughton Island	98	14	79	5	-	-	-	-	-	
Cambridge Bay	30	13	14	2	1	-	-	-	-	
Cape Dorset	26	2	24	-	-	-	-	-	-	
Chesterfield Inlet	28	16	12	-	-	-	-	-	-	
Clyde River	358	145	207	6	-	-	-	-	-	
Colville Lake	2	1	1	-	-	-	-	-	-	
Coppermine	40	27	13	-	-	-	-	-	-	
Coral Harbour	20	2	18	-	-	-	-	-	-	
Creswell Bay	4	3	1	-	-	-	-	-	-	
Detah	17	12	5	-	-	-	-	-	-	
Eskimo Point	27	8	19	-	-	-	-	-	-	
Fort Franklin	211	159	51	1	-	-	-	-	-	
Fort Good Hope	361	343	17	1	-	-	-	-	-	
Fort Liard	35	32	3	-	-	-	-	-	-	
Fort Mcpherson	27	26	1	-	-	-	-	-	-	
Fort Norman	21	15	6	-	-	-	-	-	-	
Fort Providence	38	28	10	-	-	-	-	-	-	
Fort Resolution	20	19	1	-	-	-	-	-	-	
Fort Simpson	18	18	-	-	-	-	-	-	-	
Fort Smith	20	15	5	-	-	-	-	-	-	
Frobisher Bay	38	21	17	-	-	-	-	-	-	
Gjoa Haven	23	11	12	-	-	-	-	-	-	
Grise Fiord	20	6	14	-	-	-	-	-	-	
Hall Beach	22	8	14	-	-	-	-	-	-	
Hay River	14	12	2	-	-	-	-	-	-	
Holman Island	43	6	37	-	-	-	-	-	-	
Igloolik	313	80	220	9	1	3	-	-	-	

TABLE 2
TABLEAU 2

NORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Inuvik	99	71	28	-	-	-	-	-	-	
Kakisa Lake	13	9	4	-	-	-	-	-	-	
Lac La Martre	10	5	5	-	-	-	-	-	-	
Lake Harbour	22	7	15	-	-	-	-	-	-	
Nahanni Butte	11	11	-	-	-	-	-	-	-	
Nanisivik	163	125	38	-	-	-	-	-	-	
Norman Wells	17	17	-	-	-	-	-	-	-	
Pangnirtung	25	4	21	-	-	-	-	-	-	
Paulatuk	19	12	7	-	-	-	-	-	-	
Pelly Bay	25	15	10	-	-	-	-	-	-	
Pine Point	6	5	1	-	-	-	-	-	-	
Pond Inlet	26	8	18	-	-	-	-	-	-	
Port Burwell	23	3	20	-	-	-	-	-	-	
Rae Edzo	60	43	17	-	-	-	-	-	-	
Rae Lakes	9	5	3	1	-	-	-	-	-	
Rankin Inlet	32	16	16	-	-	-	-	-	-	
Repulse Bay	24	16	8	-	-	-	-	-	-	
Resolute Bay	120	53	65	1	1	-	-	-	-	
Sachs Harbour	34	28	6	-	-	-	-	-	-	
Sanikiluaq	43	6	35	2	-	-	-	-	-	
Snowdrift	46	32	14	-	-	-	-	-	-	
Spence Bay	20	8	12	-	-	-	-	-	-	
Trout Lake	2	-	2	-	-	-	-	-	-	
Tuktoyaktuk	380	262	115	3	-	-	-	-	-	
Umingmaktak	8	8	-	-	-	-	-	-	-	
Whale Cove	23	16	7	-	-	-	-	-	-	
Wrigley	41	29	12	-	-	-	-	-	-	
Yellowknife	182	175	7	-	-	-	-	-	-	
Total	3827	2273	1515	33	3	3	-	-	-	

TABLE 2
TABLEAU 2

YUKON Community Collectivité	Total Tests Total des analyses	< 20	20-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Burwash Landing	26	26	-	-	-	-	-	-	-	
Carcross	29	29	-	-	-	-	-	-	-	
Carmacks	62	59	3	-	-	-	-	-	-	
Champagne	10	10	-	-	-	-	-	-	-	
Dawson	67	67	-	-	-	-	-	-	-	
Destruction Bay	15	15	-	-	-	-	-	-	-	
Elsa	43	42	1	-	-	-	-	-	-	
Faro	25	25	-	-	-	-	-	-	-	
Haines Junction	52	52	-	-	-	-	-	-	-	
Keno City	1	1	-	-	-	-	-	-	-	
Mayo	41	41	-	-	-	-	-	-	-	
Old Crow	78	78	-	-	-	-	-	-	-	
Pelly Crossing	26	26	-	-	-	-	-	-	-	
Ross River	31	31	-	-	-	-	-	-	-	
Tagish	1	1	-	-	-	-	-	-	-	
Teslin	31	30	1	-	-	-	-	-	-	
Watson Lake	77	77	-	-	-	-	-	-	-	
Whitehorse	247	245	2	-	-	-	-	-	-	
Total	862	855	7	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

RESULTS JANUARY 1, 1983 TO DECEMBER 31, 1996 - BY REGION, COMMUNITY, YEAR AND LEVEL
RÉSULTATS - DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996 - PAR RÉGION, COLLECTIVITÉ, ANNÉE ET NIVEAU

QUEBEC - QUÉBEC 1994

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Pointe Bleue	17	14	2	1	-	-	-	-	-	-	-	-	-	-	-	-	
Total	17	14	2	1	-	-	-	-	-	-	-	-	-	-	-	-	

QUEBEC - QUÉBEC 1990

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Caughnawaga (Kahnawake)	53	53	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	53	53	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

QUEBEC - QUÉBEC 1989

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Bécancour (Wolinak)	48	48	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Bersimis (Betsiamites)	200	196	4	-	-	-	-	-	-	-	-	-	-	-	-	-	
Escoumins (Les Escoumins)	74	69	3	-	-	1	1	-	-	-	-	-	-	-	-	-	
Odanak	54	53	-	1	-	-	-	-	-	-	-	-	-	-	-	-	
Lorette (Village Huron)	102	100	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Weymontachie	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	479	467	9	1	-	1	1	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

QUEBEC - QUÉBEC 1988

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Abitibi Dominion (Pikogan)	78	76	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Kipawa	74	71	2	1	-	-	-	-	-	-	-	-	-	-	-	-	
Lac Simon	101	86	12	2	1	-	-	-	-	-	-	-	-	-	-	-	
Maliotenam	76	73	3	-	-	-	-	-	-	-	-	-	-	-	-	-	
Maria	76	75	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Mingan	75	67	7	1	-	-	-	-	-	-	-	-	-	-	-	-	
Natashquan	76	71	4	-	1	-	-	-	-	-	-	-	-	-	-	-	
Notre-Dame-du-Nord(Timiskaming)	70	70	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pointe Bleue	121	109	9	1	2	-	-	-	-	-	-	-	-	-	-	-	
Restigouche	132	128	2	1	1	-	-	-	-	-	-	-	-	-	-	-	
Romaine	100	82	13	3	2	-	-	-	-	-	-	-	-	-	-	-	
Schefferville	77	60	9	3	2	2	-	1	-	-	-	-	-	-	-	-	
Sept-Iles	76	69	4	1	1	1	-	-	-	-	-	-	-	-	-	-	
St. Augustin	77	62	10	4	1	-	-	-	-	-	-	-	-	-	-	-	
Winneway (Longue-Pointe)	53	47	4	1	1	-	-	-	-	-	-	-	-	-	-	-	
Total	1262	1146	82	18	12	3	-	1	-	-	-	-	-	-	-	-	

QUEBEC - QUÉBEC 1986

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Maniwaki (River Desert)	101	101	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Manouan	117	106	9	1	1	-	-	-	-	-	-	-	-	-	-	-	
Obedjinan	111	63	26	9	9	1	-	2	-	1	-	-	-	-	-	-	
Weymontachie	107	105	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	436	375	37	10	10	1	-	2	-	1	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

QUEBEC - QUÉBEC 1984

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Obedjinan	6	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	6	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

QUEBEC - QUÉBEC 1983

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Obedjinan	17	14	2	1	-	-	-	-	-	-	-	-	-	-	-	-	
Pointe Bleue	8	8	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	25	22	2	1	-	-	-	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

ONTARIO 1996

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	175	150	17	5	2	1	-	-	-	-	-	-	-	-	-	-	
Whitedog	77	76	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	252	226	18	5	2	1	-	-	-	-	-	-	-	-	-	-	

ONTARIO 1995

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	117	108	7	1	1	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	56	53	2	1	-	-	-	-	-	-	-	-	-	-	-	-	
Total	173	161	9	2	1	-	-	-	-	-	-	-	-	-	-	-	

ONTARIO 1994

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	17	13	2	2	-	-	-	-	-	-	-	-	-	-	-	-	
Total	21	17	2	2	-	-	-	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

ONTARIO 1993

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	78	61	8	5	2	2	-	-	-	-	-	-	-	-	-	-	
Whitedog	50	44	5	1	-	-	-	-	-	-	-	-	-	-	-	-	
Total	128	105	13	6	2	2	-	-	-	-	-	-	-	-	-	-	

ONTARIO 1992

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Dalles	1	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	
Grassy Narrows	182	147	17	7	3	4	3	1	-	-	-	-	-	-	-	-	
Northwest Angle 33	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	
Whitedog	37	35	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	221	182	19	7	3	5	4	1	-	-	-	-	-	-	-	-	

ONTARIO 1991

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	9	7	1	1	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	79	74	5	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	88	81	6	1	-	-	-	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

ONTARIO 1990

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Angle Inlet	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Grassy Narrows	56	49	2	2	2	1	-	-	-	-	-	-	-	-	-	-	
Rat Portage	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	105	91	8	6	-	-	-	-	-	-	-	-	-	-	-	-	
Whitefish Bay	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	164	143	10	8	2	1	-	-	-	-	-	-	-	-	-	-	

ONTARIO 1989

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Cape Crocker	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Dalles	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Grassy Narrows	129	100	12	6	4	2	-	1	1	1	2	-	-	-	-	-	
Northwest Angle 33	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Rat Portage	3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal Lake	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Washagamis Bay	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	340	314	13	5	3	2	3	-	-	-	-	-	-	-	-	-	
Whitefish Bay	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	485	430	25	11	7	4	3	1	1	1	2	-	-	-	-	-	

TABLE 3
TABLEAU 3

ONTARIO 1988

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Angle Inlet	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Bear Island	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Grassy Narrows	136	103	12	4	6	3	5	-	1	-	2	-	-	-	-	-	
Northwest Angle 33	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Rat Portage	5	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal Lake	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	10	10	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitefish Bay	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	170	137	12	4	6	3	5	-	1	-	2	-	-	-	-	-	

ONTARIO 1987

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	131	99	13	3	3	4	2	4	-	-	3	-	-	-	-	-	
Mcdiarmid	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Northwest Angle 33	5	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pickle Lake	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Rat Portage	3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal Lake	8	8	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Washagamis Bay	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	213	186	14	4	5	1	1	1	-	1	-	-	-	-	-	-	
Whitefish Bay	9	9	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	372	313	27	7	8	5	3	5	-	1	3	-	-	-	-	-	

TABLE 3
TABLEAU 3

ONTARIO 1986

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	68	39	7	6	4	2	4	2	1	1	2	-	-	-	-	-	
Rat Portage	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal Lake	7	7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Washagamis Bay	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	282	214	30	15	11	5	2	1	1	2	1	-	-	-	-	-	
Whitefish Bay	7	7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	369	272	37	21	15	7	6	3	2	3	3	-	-	-	-	-	

ONTARIO 1985

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Bear Island	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Grassy Narrows	83	51	8	5	6	2	3	1	-	3	4	-	-	-	-	-	
Gull Bay	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Northwest Angle 33	3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Rat Portage	6	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Sabaskong	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal Lake	7	7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Washagamis Bay	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	278	226	22	12	5	5	2	2	3	-	1	-	-	-	-	-	
Whitefish Bay	11	11	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	393	309	30	17	11	7	5	3	3	3	5	-	-	-	-	-	

TABLE 3
TABLEAU 3

ONTARIO 1984

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Grassy Narrows	128	85	8	12	6	3	2	4	-	3	5	-	-	-	-	-	
Rat Portage	14	13	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Sabaskong	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal Lake	7	7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Washagamis Bay	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Whitedog	363	285	40	10	10	8	5	2	1	1	1	-	-	-	-	-	
Whitefish Bay	11	11	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	525	403	49	22	16	11	7	6	1	4	6	-	-	-	-	-	

TABLE 3
TABLEAU 3

ONTARIO 1983

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Angling Lake	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Bearskin Lake	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Big Trout Lake	13	12	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Cat Lake	11	11	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Collins	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Deer Lake	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Fort Hope	10	10	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Fort Severn	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Frenchmans Head	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Grassy Narrows	170	130	8	4	5	9	3	2	3	1	5	-	-	-	-	-	
Kasabonika	3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Kingfisher Lake	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Lansdowne House	9	7	1	1	-	-	-	-	-	-	-	-	-	-	-	-	
Muskrat Dam	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
New Osnaburgh	6	4	1	1	-	-	-	-	-	-	-	-	-	-	-	-	
North Spirit Lake	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Northwest Angle 33	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Ogoki Post	100	63	21	7	5	1	-	2	1	-	-	-	-	-	-	-	
Pikangikum	26	26	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Poplar Hill	7	4	2	-	1	-	-	-	-	-	-	-	-	-	-	-	
Rat Portage	21	21	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Round Lake (Weagamow Lake)	8	8	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Sachigo Lake	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Sandy Lake - Ontario	15	14	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal Lake	10	10	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Slate Falls	3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Summer Beaver	6	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Washagamis Bay	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Webequie	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
WhiteDog	350	253	37	18	14	12	9	5	1	-	1	-	-	-	-	-	
Whitefish Bay	8	8	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Wunnumin	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	828	645	72	31	25	22	12	9	5	1	6	-	-	-	-	-	

TABLE 3
TABLEAU 3

MANITOBA 1989

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Cross Lake	504	487	11	1	2	-	-	-	1	-	1	1	-	-	-	-	
Fox Lake (Manitoba)	24	18	3	1	-	-	-	-	-	-	2	-	-	-	-	-	
God's Lake	3	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
God's River	37	33	4	-	-	-	-	-	-	-	-	-	-	-	-	-	
Granville Lake	4	3	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Lac Brochet	37	35	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Nelson House	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Norway House	117	112	4	1	-	-	-	-	-	-	-	-	-	-	-	-	
Pukatawagan	8	3	2	-	2	1	-	-	-	-	-	-	-	-	-	-	
Shamattawa	20	18	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Split Lake	208	205	3	-	-	-	-	-	-	-	-	-	-	-	-	-	
Tadoule Lake	8	6	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
York Landing	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	972	924	35	3	4	1	-	-	1	-	3	1	-	-	-	-	

TABLE 3
TABLEAU 3

MANITOBA 1988

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Cross Lake	10	10	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Granville Lake	6	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Lac Brochet	11	9	1	-	-	-	-	-	-	1	-	-	-	-	-	-	
Nelson House	527	471	37	12	4	3	-	-	-	-	-	-	-	-	-	-	
Norway House	648	625	16	6	1	-	-	-	-	-	-	-	-	-	-	-	
Pauingassi	42	42	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal River	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
South Indian Lake	347	271	40	16	14	5	-	-	-	1	-	-	-	-	-	-	
St. Theresa Point	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
York Landing	103	100	2	-	1	-	-	-	-	-	-	-	-	-	-	-	
Total	1696	1536	96	34	20	8	-	-	-	2	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

MANITOBA 1987

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Bloodvein	1	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Cross Lake	8	6	-	1	-	-	1	-	-	-	-	-	-	-	-	-	
Easterville	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
God's Lake	1	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	
Granville Lake	5	4	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Lac Brochet	12	9	1	2	-	-	-	-	-	-	-	-	-	-	-	-	
Little Grand Rapids	6	3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	
Nelson House	106	90	8	4	3	1	-	-	-	-	-	-	-	-	-	-	
Pauingassi	15	11	1	2	1	-	-	-	-	-	-	-	-	-	-	-	
Pukatawagan	30	13	11	4	2	-	-	-	-	-	-	-	-	-	-	-	
Red Sucker Lake	2	-	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Sioux Valley	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
St. Theresa Point	4	1	-	1	1	-	1	-	-	-	-	-	-	-	-	-	
The Pas	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	193	140	28	15	7	1	2	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

MANITOBA 1986

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Bloodvein	43	39	3	-	-	-	-	1	-	-	-	-	-	-	-	-	
Chemahawin (Easterville)	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Cross Lake	6	2	3	1	-	-	-	-	-	-	-	-	-	-	-	-	
Dakota Plains	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Fisher River	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Fort Alexander	40	37	3	-	-	-	-	-	-	-	-	-	-	-	-	-	
Fox Lake (Manitoba)	8	5	1	1	1	-	-	-	-	-	-	-	-	-	-	-	
Gods Lake	29	15	8	5	1	-	-	-	-	-	-	-	-	-	-	-	
Hollow Water	2	1	-	-	-	-	-	-	1	-	-	-	-	-	-	-	
Ilford	10	9	-	-	1	-	-	-	-	-	-	-	-	-	-	-	
Jackhead	3	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Lac Brochet	19	15	3	1	-	-	-	-	-	-	-	-	-	-	-	-	
Moose Lake	1	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Nelson House	328	250	48	17	9	2	1	-	-	1	-	-	-	-	-	-	
Norway House	76	74	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pine Creek	3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pukatawagan	16	4	7	4	1	-	-	-	-	-	-	-	-	-	-	-	
Shamattawa	6	2	3	1	-	-	-	-	-	-	-	-	-	-	-	-	
Shoal River	67	67	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
South Indian Lake	18	9	2	6	-	-	1	-	-	-	-	-	-	-	-	-	
Split Lake	102	81	13	4	3	-	-	-	1	-	-	-	-	-	-	-	
St. Theresa Point	8	3	3	2	-	-	-	-	-	-	-	-	-	-	-	-	
Swan Lake	15	15	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Tadoule Lake	6	2	-	2	1	-	1	-	-	-	-	-	-	-	-	-	
The Pas	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
York Landing	61	47	5	5	1	-	-	-	-	-	3	-	-	-	-	-	
Total	897	712	106	49	18	2	3	1	2	1	3	-	-	-	-	-	

TABLE 3
TABLEAU 3

MANITOBA 1985

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Brokenhead	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Chemahawin (Easterville)	43	42	-	1	-	-	-	-	-	-	-	-	-	-	-	-	
Cross Lake	109	105	1	2	-	1	-	-	-	-	-	-	-	-	-	-	
Fox Lake (Manitoba)	2	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
God's Lake	21	16	3	-	2	-	-	-	-	-	-	-	-	-	-	-	
Granville Lake	12	10	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Hollow Water	34	31	1	1	-	-	-	1	-	-	-	-	-	-	-	-	
Lac Brochet	2	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Lake St. Martin	5	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Little Black River	21	21	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Little Grand Rapids	5	3	1	1	-	-	-	-	-	-	-	-	-	-	-	-	
Moose Lake	33	30	3	-	-	-	-	-	-	-	-	-	-	-	-	-	
Nelson House	133	103	16	5	7	1	-	1	-	-	-	-	-	-	-	-	
Oxford House	8	8	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pauingassi	7	-	3	1	2	-	-	-	-	-	1	-	-	-	-	-	
Pine Creek	53	50	1	2	-	-	-	-	-	-	-	-	-	-	-	-	
Pukatawagan	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Red Sucker Lake	7	1	3	1	1	1	-	-	-	-	-	-	-	-	-	-	
Shamattawa	10	4	3	3	-	-	-	-	-	-	-	-	-	-	-	-	
South Indian Lake	64	34	17	9	2	-	2	-	-	-	-	-	-	-	-	-	
Split Lake	69	54	8	4	3	-	-	-	-	-	-	-	-	-	-	-	
St. Theresa Point	129	120	7	1	-	-	1	-	-	-	-	-	-	-	-	-	
Sypsumville	25	25	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
The Pas	127	125	1	-	-	-	1	-	-	-	-	-	-	-	-	-	
Valley River	20	19	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Wasagamack	29	22	5	1	-	-	1	-	-	-	-	-	-	-	-	-	
Waywayseecappo	26	26	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
York Landing	40	35	2	1	1	-	-	-	-	-	1	-	-	-	-	-	
Total	1037	894	80	33	18	3	5	2	-	-	2	-	-	-	-	-	

TABLE 3
TABLEAU 3

MANITOBA 1984

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Brochet	33	31	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Cross Lake	214	206	6	2	-	-	-	-	-	-	-	-	-	-	-	-	
Fox Lake (Manitoba)	32	20	4	3	2	1	2	-	-	-	-	-	-	-	-	-	
God's Lake	106	77	26	2	1	-	-	-	-	-	-	-	-	-	-	-	
God's River	5	2	1	1	1	-	-	-	-	-	-	-	-	-	-	-	
Lac Brochet	29	24	4	1	-	-	-	-	-	-	-	-	-	-	-	-	
Little Grand Rapids	5	4	-	-	-	1	-	-	-	-	-	-	-	-	-	-	
Nelson House	268	226	21	11	4	3	1	-	-	1	1	-	-	-	-	-	
Norway House	219	210	5	2	1	1	-	-	-	-	-	-	-	-	-	-	
Pauingassi	16	6	-	4	1	1	1	1	-	-	2	-	-	-	-	-	
Peguis	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pukatawagan	52	29	11	11	1	-	-	-	-	-	-	-	-	-	-	-	
Red Sucker Lake	24	16	6	-	1	1	-	-	-	-	-	-	-	-	-	-	
Shamattawa	44	36	6	2	-	-	-	-	-	-	-	-	-	-	-	-	
South Indian Lake	105	68	20	10	4	3	-	-	-	-	-	-	-	-	-	-	
Split Lake	105	93	11	-	1	-	-	-	-	-	-	-	-	-	-	-	
Tadoule Lake	28	17	2	1	4	-	3	1	-	-	-	-	-	-	-	-	
York Landing	42	27	11	3	-	-	-	1	-	-	-	-	-	-	-	-	
Total	1329	1094	136	53	21	11	7	3	-	1	3	-	-	-	-	-	

TABLE 3
TABLEAU 3

MANITOBA 1983

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Brokenhead	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Cross Lake	7	7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Fairford	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
God's Lake	24	18	3	3	-	-	-	-	-	-	-	-	-	-	-	-	
God's River	7	1	5	1	-	-	-	-	-	-	-	-	-	-	-	-	
Grand Rapids	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Granville Lake	6	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Lac Brochet	4	3	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Little Grand Rapids	10	8	1	1	-	-	-	-	-	-	-	-	-	-	-	-	
Nelson House	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Norway House	52	52	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pukatawagan	73	51	9	9	1	2	1	-	-	-	-	-	-	-	-	-	
Red Sucker Lake	5	4	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Shamattawa	15	13	-	1	1	-	-	-	-	-	-	-	-	-	-	-	
South Indian Lake	53	50	-	3	-	-	-	-	-	-	-	-	-	-	-	-	
Split Lake	9	9	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
York Landing	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	286	243	20	18	2	2	1	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

SASKATCHEWAN 1989

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Witchekan Lake	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

SASKATCHEWAN 1988

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Sandy Bay	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

SASKATCHEWAN 1984

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Chitek Lake (Pelican Lake)	20	20	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	20	20	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

SASKATCHEWAN 1983

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Chitek Lake (Pelican Lake)	33	32	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	33	32	1	-	-	-	-	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

ALBERTA 1989

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Fox Lake	6	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Peigan	161	161	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	167	167	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE 1995

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Fort Babine	28	28	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Tachet	21	21	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Woyenne	71	71	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	120	120	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE 1992

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Burns Lake	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Cheslatta	35	35	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Nee-Tahi-Buhn	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	38	38	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE 1991

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Fort Ware	88	86	2	-	-	-	-	-	-	-	-	-	-	-	-	-	
Ingenika	93	82	7	2	-	-	2	-	-	-	-	-	-	-	-	-	
Mount Currie	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Pinchi & Tachi	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	183	170	9	2	-	-	2	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE 1986

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Pinchi & Tachi	16	15	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	16	15	1	-	-	-	-	-	-	-	-	-	-	-	-	-	

BRITISH COLUMBIA - COLOMBIE BRITANNIQUE 1984

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Squamish	8	8	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	8	8	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

TABLE 3
TABLEAU 3

NORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST 1989

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Clyde River	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	4	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

NORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST 1988

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Clyde River	20	18	1	-	-	1	-	-	-	-	-	-	-	-	-	-	
Total	20	18	1	-	-	1	-	-	-	-	-	-	-	-	-	-	

NORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST 1987

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Clyde River	72	20	14	11	7	6	5	4	2	1	2	-	-	-	-	-	
Pond Inlet	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	73	21	14	11	7	6	5	4	2	1	2	-	-	-	-	-	

TABLE 3
TABLEAU 3

ORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST 1986

ommunity ollectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
lyde River	18	13	3	2	-	-	-	-	-	-	-	-	-	-	-	-	
Total	18	13	3	2	-	-	-	-	-	-	-	-	-	-	-	-	

ORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST 1985

ommunity ollectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
lyde River	28	23	2	-	2	1	-	-	-	-	-	-	-	-	-	-	
gloolik	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
esolute Bay	32	11	9	7	4	1	-	-	-	-	-	-	-	-	-	-	
Total	62	36	11	7	6	2	-	-	-	-	-	-	-	-	-	-	

ORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST 1984

ommunity ollectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
roughton Island	70	11	8	14	13	5	7	4	3	1	4	-	-	-	-	-	
lyde River	18	12	1	2	1	2	-	-	-	-	-	-	-	-	-	-	
reswell Bay	4	3	1	-	-	-	-	-	-	-	-	-	-	-	-	-	
gloolik	4	-	3	-	1	-	-	-	-	-	-	-	-	-	-	-	
anisivik	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
ond Inlet	2	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	
esolute Bay	66	40	7	7	6	1	3	1	-	-	-	1	-	-	-	-	
Total	165	68	20	23	21	8	11	5	3	1	4	1	-	-	-	-	

TABLE 3
TABLEAU 3

NORTHWEST TERRITORIES - TERRITOIRES DU NORD-OUEST 1983

Community Collectivité	Total Tests Total des analyses	< 20	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Clyde River	25	9	7	8	-	1	-	-	-	-	-	-	-	-	-	-	
Fort Franklin	22	16	2	-	-	1	2	1	-	-	-	-	-	-	-	-	
Pond Inlet	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Tuktoyaktuk	20	13	4	-	2	1	-	-	-	-	-	-	-	-	-	-	
Wrigley	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Total	69	40	13	8	2	3	2	1	-	-	-	-	-	-	-	-	

TABLE 4
TABLEAU 4

TOTAL INDIVIDUALS > 100 ppb JANUARY 1,1983 TO DECEMBER 31,1996 - BY REGION AND LEVEL
NOMBRE TOTAL DE PERSONNES > 100 µg/L DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996 - PAR RÉGION ET NIVEAU

() Percentage of Total
() Pourcentage du total

Region Région	Total Individuals >100 ppb Nbre total de personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
ATLANTIC ATLANTIQUE	0	-	-	-	-	-	-	
QUEBEC QUÉBEC	0	-	-	-	-	-	-	
ONTARIO	14	14 (100.00)	-	-	-	-	-	
MANITOBA	7	6 (85.71)	1 (14.29)	-	-	-	-	
SASKATCHEWAN	0	-	-	-	-	-	-	
ALBERTA	0	-	-	-	-	-	-	
BRITISH COLUMBIA COLOMBIE BRITANNIQUE	0	-	-	-	-	-	-	
NORTHWEST TERRITORIES TERRITOIRES DU NORD-OUEST	7	6 (85.71)	1 (14.29)	-	-	-	-	
YUKON	0	-	-	-	-	-	-	
Total	28	26 (92.86)	2 (7.14)	-	-	-	-	

TABLE 4A
TABLEAU 4A

TOTAL INDIVIDUALS > 100 ppb TO DECEMBER 31, 1996 - BY REGION AND LEVEL
NOMBRE TOTAL DE PERSONNES > 100 µg/L AU 31 DÉCEMBRE 1996 - PAR RÉGION ET NIVEAU

() Percentage of Total
() Pourcentage du total

Region Région	Total Individuals >100 ppb Nbre total de personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
ATLANTIC ATLANTIQUE	0	-	-	-	-	-	-	
QUEBEC QUÉBEC	422	379 (89.81)	32 (7.58)	7 (1.66)	1 (0.24)	2 (0.47)	1 (0.24)	
ONTARIO	131	105 (80.15)	16 (12.21)	3 (2.29)	5 (3.82)	-	2 (1.53)	
MANITOBA	19	17 (89.47)	2 (10.53)	-	-	-	-	
SASKATCHEWAN	5	5 (100.00)	-	-	-	-	-	
ALBERTA	1	1 (100.00)	-	-	-	-	-	
BRITISH COLUMBIA COLOMBIE BRITANNIQUE	3	3 (100.00)	-	-	-	-	-	
NORTHWEST TERRITORIES TERRITOIRES DU NORD-OUEST	34	28 (82.36)	3 (8.82)	3 (8.82)	-	-	-	
YUKON	0	-	-	-	-	-	-	
Total	615	538 (87.48)	53 (8.62)	13 (2.11)	6 (0.98)	2 (0.32)	3 (0.49)	

TABLE 5
TABLEAU 5

TOTAL INDIVIDUALS > 100 ppb JANUARY 1,1983 TO DECEMBER 31, 1996 -
BY REGION, COMMUNITY AND LEVEL

NOMBRE TOTAL DE PERSONNES > 100 µg/L DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996 -
PAR RÉGION, COLLECTIVITÉ ET NIVEAU

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-	
Quebec Québec		0	-	-	-	-	-	-	
Ontario	Grassy Narrows	11	11	-	-	-	-	-	
	Whitedog	3	3	-	-	-	-	-	
Manitoba	Cross Lake	2	1	1	-	-	-	-	
	Fox Lake (Manitoba)	2	2	-	-	-	-	-	
	Nelson House	1	1	-	-	-	-	-	
	Pauingassi	1	1	-	-	-	-	-	
	York Landing	1	1	-	-	-	-	-	
Saskatchewan		0	-	-	-	-	-	-	
Alberta		0	-	-	-	-	-	-	
British Columbia Colombie Britanique		0	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest	Broughton Island	4	4	-	-	-	-	-	
	Clyde River	2	2	-	-	-	-	-	
	Resolute Bay	1	-	1	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 5A
TABLEAU 5A

TOTAL INDIVIDUALS > 100 ppb TO DECEMBER 31, 1996 -
BY REGION, COMMUNITY AND LEVEL

NOMBRE TOTAL DE PERSONNES > 100 ug/L AU 31 DÉCEMBRE 1996 -
PAR RÉGION, COLLECTIVITÉ ET NIVEAU

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-	
Quebec Québec	Abitibi Dominion (Pikogan)	4	2	-	2	-	-	-	
	Fort George	26	22	3	1	-	-	-	
	Inukjuaq (Unukjuaq)	15	15	-	-	-	-	-	
	Ivujivik	7	7	-	-	-	-	-	
	Kangirsujak (Wakeham Bay)	2	2	-	-	-	-	-	
	Kujuarapik (Great Whale)	52	43	8	-	1	-	-	
	Kuujjuaq (Fort Chimo)	1	1	-	-	-	-	-	
	Lac Simon	6	5	1	-	-	-	-	
	Maniwaki (River Desert)	1	1	-	-	-	-	-	
	Manouan	4	4	-	-	-	-	-	
	Mistassini	155	144	10	1	-	-	-	
	Obedjinah	11	10	1	-	-	-	-	
	Paint Hills (Wemindji)	4	3	-	1	-	-	-	
	Puvirnituq (Povungnituk)	1	1	-	-	-	-	-	
	Quartaq (Koartak)	8	8	-	-	-	-	-	
	Rupert House (Waskagonish)	5	5	-	-	-	-	-	
	Salluit (Sugluk)	50	45	5	-	-	-	-	
	Schefferville	17	16	1	-	-	-	-	
	Waswanipi	53	45	3	2	-	2	1	

TABLE 5A
TABLEAU 5A

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Ontario	Attawapiskat	1	1	-	-	-	-	-	
	Deer Lake	6	6	-	-	-	-	-	
	Eagle Lake	1	1	-	-	-	-	-	
	Grassy Narrows	70	50	12	2	5	-	1	
	North Spirit Lake	2	2	-	-	-	-	-	
	Ogoki Post	1	1	-	-	-	-	-	
	Poplar Hill	7	6	1	-	-	-	-	
	Whitedog	43	38	3	1	-	-	1	
Manitoba	Cross Lake	2	1	1	-	-	-	-	
	Dakota Plains	1	-	1	-	-	-	-	
	Fox Lake (Manitoba)	2	2	-	-	-	-	-	
	Lac Brochet	1	1	-	-	-	-	-	
	Nelson House	2	2	-	-	-	-	-	
	Pauingassi	4	4	-	-	-	-	-	
	South Indian Lake	6	6	-	-	-	-	-	
	York Landing	1	1	-	-	-	-	-	
Saskatchewan	Kahkewistahaw	1	1	-	-	-	-	-	
	Kinoosao	1	1	-	-	-	-	-	
	Lac La Ronge	1	1	-	-	-	-	-	
	Little Black Bear	1	1	-	-	-	-	-	
	Mistawasis	1	1	-	-	-	-	-	
Alberta	Blood	1	1	-	-	-	-	-	

TABLE 5A
TABLEAU 5A

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
British Columbia	Necoslie	1	1	-	-	-	-	-	
Colombie-	Nimpkish	1	1	-	-	-	-	-	
Britanique	Sheshaht (Tsahaheh)	1	1	-	-	-	-	-	
Northwest	Arctic Bay	1	1	-	-	-	-	-	
Territories	Broughton Island	4	4	-	-	-	-	-	
Territoires du	Cambridge Bay	2	1	1	-	-	-	-	
Nord-Ouest	Clyde River	5	5	-	-	-	-	-	
	Fort Franklin	1	1	-	-	-	-	-	
	Fort Good Hope	1	1	-	-	-	-	-	
	Igloolik	13	9	1	3	-	-	-	
	Rae Lakes	1	1	-	-	-	-	-	
	Resolute Bay	2	1	1	-	-	-	-	
	Sanikiluaq	2	2	-	-	-	-	-	
	Tuktoyaktuk	2	2	-	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 6
TABLEAU 6

TOTAL INDIVIDUALS > 100 ppb TO DECEMBER 31, 1996 - BY REGION AND YEAR
NOMBRE TOTAL DE PERSONNES > 100 µg/L AU 31 DÉCEMBRE 1996 - PAR RÉGION ET ANNÉE

Region Région	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983
Atlantic Atlantique	0	0	0	0	0	0	0	0	0	0	0	0	0
Quebec Québec	20	2	0	0	131	95	85	169	13	6	1	0	0
Ontario	32	0	18	9	47	19	7	21	22	8	4	7	6
Manitoba	0	0	0	2	0	2	2	1	8	0	0	1	0
Saskatchewan	0	0	0	0	0	0	0	5	0	0	0	0	0
Alberta	0	0	0	0	0	0	1	0	0	0	0	0	0
British Columbia Colombie Britanique	0	0	0	0	0	0	1	2	0	0	0	0	0
Northwest Territories Territoires du Nord-Ouest	13	1	0	0	0	3	4	6	1	1	1	0	0
Yukon	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	65	3	18	11	178	119	100	204	44	15	6	8	6

TABLE 6
TABLEAU 6

Region Région	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
Atlantic Atlantique	0	0	0	0	0	0	0	0	0	0	0	0	0
Quebec Québec	0	0	0	0	0	0	0	0	0	0	0	0	0
Ontario	5	4	3	2	2	2	0	0	0	0	0	0	0
Manitoba	2	2	1	0	0	4	0	0	0	0	0	0	0
Saskatchewan	0	0	0	0	0	0	0	0	0	0	0	0	0
Alberta	0	0	0	0	0	0	0	0	0	0	0	0	0
British Columbia Colombie Britanique	0	0	0	0	0	0	0	0	0	0	0	0	0
Northwest Territories Territoires du Nord-Ouest	5	0	0	2	0	0	0	0	0	0	0	0	0
Yukon	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	12	6	4	4	2	6	0	0	0	0	0	0	0

TABLE 7
TABLEAU 7

TOTAL INDIVIDUALS > 100 ppb JANUARY 1, 1983 TO DECEMBER 31, 1996 - BY REGION, COMMUNITY, YEAR AND LEVEL
NOMBRE TOTAL DE PERSONNES > 100 µg/L DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996 -
PAR RÉGION, COLLECTIVITÉ, ANNÉE ET NIVEAU

1989

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-	
Quebec Québec		0	-	-	-	-	-	-	
Ontario	Grassy Narrows	2	2	-	-	-	-	-	
Manitoba	Cross Lake	2	1	1	-	-	-	-	
	Fox Lake (Manitoba)	2	2	-	-	-	-	-	
Saskatchewan		0	-	-	-	-	-	-	
Alberta		0	-	-	-	-	-	-	
British Columbia Colombie Britanique		0	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest		0	-	-	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 7
TABLEAU 7

1988

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-	
Quebec Québec		0	-	-	-	-	-	-	
Ontario	Grassy Narrows	2	2	-	-	-	-	-	
Manitoba		0	-	-	-	-	-	-	
Saskatchewan		0	-	-	-	-	-	-	
Alberta		0	-	-	-	-	-	-	
British Columbia Colombie Britanique		0	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest		0	-	-	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 7
TABLEAU 7

1987

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-	
Quebec Québec		0	-	-	-	-	-	-	
Ontario	Grassy Narrows	2	2	-	-	-	-	-	
Manitoba		0	-	-	-	-	-	-	
Saskatchewan		0	-	-	-	-	-	-	
Alberta		0	-	-	-	-	-	-	
British Columbia Colombie Britanique		0	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest	Clyde River	2	2	-	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 7
TABLEAU 7

1986

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0		-	-	-	-	-	
Quebec Québec		0	-	-	-	-	-	-	
Ontario	Grassy Narrows	2	2	-	-	-	-	-	
	Whitedog	1	1	-	-	-	-	-	
Manitoba	York Landing	1	1	-	-	-	-	-	
Saskatchewan		0	-	-	-	-	-	-	
Alberta		0	-	-	-	-	-	-	
British Columbia Colombie Britanique		0	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest		0	-	-	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 7
TABLEAU 7

1985

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-	
Quebec Québec		0	-	-	-	-	-	-	
Ontario	Grassy Narrows	3	3	-	-	-	-	-	
	Whitedog	1	1	-	-	-	-	-	
Manitoba	Pauingassi	1	1	-	-	-	-	-	
	York Landing	1	1	-	-	-	-	-	
Saskatchewan		0	-	-	-	-	-	-	
Alberta		0	-	-	-	-	-	-	
British Columbia Colombie Britanique		0	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest		0	-	-	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 7
TABLEAU 7

1984

Region Région	Community Collectivité	Individuals >100 ppb personnes >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-	
Quebec Québec		0	-	-	-	-	-	-	
Ontario	Grassy Narrows	4	4	-	-	-	-	-	
	Whitedog	1	1	-	-	-	-	-	
Manitoba	Nelson House	1	1	-	-	-	-	-	
	Pauingassi	1	1	-	-	-	-	-	
Saskatchewan		0	-	-	-	-	-	-	
Alberta		0	-	-	-	-	-	-	
British Columbia Colombie Britanique		0	-	-	-	-	-	-	
Northwest Territories	Broughton Island	4	4	-	-	-	-	-	
Territoires du Nord-Ouest	Resolute Bay	1	-	1	-	-	-	-	
Yukon		0	-	-	-	-	-	-	

TABLE 7
TABLEAU 7

1983

Region Région	Community Collectivité	Individuals personnes	>100 ppb >100 µg/L	100-199	200-299	300-399	400-499	500-599	600-699	ppb µg/L
Atlantic Atlantique		0	-	-	-	-	-	-		
Quebec Québec		0	-	-	-	-	-	-		
Ontario	Grassy Narrows	5	5	-	-	-	-	-		
	Whitedog	1	1	-	-	-	-	-		
Manitoba		0	-	-	-	-	-	-		
Saskatchewan		0	-	-	-	-	-	-		
Alberta		0	-	-	-	-	-	-		
British Columbia Colombie Britanique		0	-	-	-	-	-	-		
Northwest Territories Territoires du Nord-Ouest		0	-	-	-	-	-	-		
Yukon		0	-	-	-	-	-	-		

TABLE 8
TABLEAU 8

TOTAL INDIVIDUALS > 100 ppb JANUARY 1, 1983 TO DECEMBER 31 1996 - BY REGION, SEX AND AGE
NOMBRE TOTAL DE PERSONNES > 100 µg/L DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996
PAR RÉGION SEXE ET AGE

Region Région	Individuals > 100 ppb Personnes > 100 µg/L	< 1 Year < 1 an		1-14 Years 1-14 ans		15-45 Years 15-45 ans		46-65 Years 46-65 ans		> 65 Years > 65 ans	
		M	F	M	F	M	F	M	F	M	F
Atlantic Atlantique	0	-	-	-	-	-	-	-	-	-	-
Quebec Québec	0	-	-	-	-	-	-	-	-	-	-
Ontario	14	-	-	-	-	11	1	2	-	-	-
Manitoba	7	-	-	1	-	1	1	-	-	3	1
Saskatchewan	0	-	-	-	-	-	-	-	-	-	-
Alberta	0	-	-	-	-	-	-	-	-	-	-
British Columbia Colombie Britannique	0	-	-	-	-	-	-	-	-	-	-
Northwest Territories Territoires du Nord-Ouest	7	-	-	-	-	6	-	1	-	-	-
Yukon	0	-	-	-	-	-	-	-	-	-	-
Total	28	-	-	1	-	18	2	3	-	3	1

TABLE 8A
TABLEAU 8A

TOTAL INDIVIDUALS > 100 ppb TO DECEMBER 31, 1996 - BY REGION, SEX AND AGE
NOMBRE TOTAL DE PERSONNES > 100 µg/L AU 31 DÉCEMBRE 1996
PAR RÉGION, SEXE ET AGE

Region Région	Individuals > 100 ppb Personnes > 100 µg/L	< 1 Year < 1 an		1-14 Years 1-14 ans		15-45 Years 15-45 ans		46-65 Years 46-65 ans		> 65 Years > 65 ans	
		M	F	M	F	M	F	M	F	M	F
Atlantic Atlantique	0	-	-	-	-	-	-	-	-	-	-
Quebec Québec	422	6	4	18	18	93	79	88	76	21	19
Ontario	131	4	1	8	10	48	19	23	7	8	3
Manitoba	19	-	-	1	-	4	4	3	2	3	2
Saskatchewan	5	-	-	-	-	-	4	-	-	1	-
Alberta	1	-	-	-	-	-	-	-	1	-	-
British Columbia Colombie Britannique	3	-	-	-	-	1	1	-	1	-	-
Northwest Territories Territoires du Nord-Ouest	34	-	2	-	2	12	11	3	4	-	-
Yukon	0	-	-	-	-	-	-	-	-	-	-
Total	615	10	7	27	30	158	118	117	91	33	24

TABLE 9
TABLEAU 9

TOTAL INDIVIDUALS > 100 ppb JANUARY 1, 1983 TO DECEMBER 31, 1996
- BY REGION, COMMUNITY, SEX, AGE AND LEVEL
NOMBRE TOTAL DE PERSONNES > 100 µg/L DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996
PAR RÉGION, COLLECTIVITÉ, SEXE, AGE ET NIVEAU

Region Région	Community Collectivité	Level (ppb) Niveau (µg/L)	< 1 Year < 1 an		1-14 Years 1-14 ans		15-45 Years 15-45 ans		46-65 Years 46-65 ans		> 65 Years > 65 ans	
			M	F	M	F	M	F	M	F	M	F
Atlantic Atlantique		0	-	-	-	-	-	-	-	-	-	-
Quebec Québec		0	-	-	-	-	-	-	-	-	-	-
Ontario	Grassy Narrows	100-199	-	-	-	-	8	1	2	-	-	-
	Whitedog	100-199	-	-	-	-	3	-	-	-	-	-
Manitoba	Cross Lake	100-199	-	-	-	-	-	-	-	-	1	-
		200-299	-	-	-	-	-	-	-	-	1	-
	Fox Lake (Manitoba)	100-199	-	-	1	-	-	-	-	-	-	1
	Nelson House	100-199	-	-	-	-	-	1	-	-	-	-
	Pauingassi	100-199	-	-	-	-	-	-	-	-	1	-
	York Landing	100-199	-	-	-	-	1	-	-	-	-	-
Saskatchewan		0	-	-	-	-	-	-	-	-	-	-
Alberta		0	-	-	-	-	-	-	-	-	-	-
British Columbia Colombie Britannique		0	-	-	-	-	-	-	-	-	-	-
Northwest Territories Territoires du Nord-Ouest	Broughton Island	100-199	-	-	-	-	4	-	-	-	-	-
	Clyde River	100-199	-	-	-	-	1	-	1	-	-	-
	Resolute Bay	200-299	-	-	-	-	1	-	-	-	-	-
Yukon		0	-	-	-	-	-	-	-	-	-	-

TABLE 9A
TABLEAU 9A

TOTAL INDIVIDUALS > 100 ppb TO DECEMBER 31, 1996
- BY REGION, COMMUNITY, SEX, AGE AND LEVEL
NOMBRE TOTAL DE PERSONNES > 100 µg/L AU 31 DÉCEMBRE 1996
PAR RÉGION, COLLECTIVITÉ, SEXE, AGE ET NIVEAU

Region Région	Community Collectivité	Level (ppb) Niveau (µg/L)	< 1 Year < 1 an		1-14 Years 1-14 ans		15-45 Years 15-45 ans		46-65 Years 46-65 ans		> 65 Years > 65 ans	
			M	F	M	F	M	F	M	F	M	F
Atlantic Atlantique		0	-	-	-	-	-	-	-	-	-	-
Quebec Québec	Abitibi Dominion	100-199	-	-	-	-	-	1	-	-	1	-
	(Pikogan)	300-399	-	-	-	-	-	-	1	1	-	-
	Fort George	100-199	-	-	-	-	6	3	6	5	2	-
		200-299	-	-	-	-	-	-	1	1	-	1
		300-399	-	-	-	-	-	-	1	-	-	-
	Inukjuaq (Unukjuaq)	100-199	-	-	-	-	6	3	5	1	-	-
	Ivujivik	100-199	1	-	-	-	1	2	2	1	-	-
	Kangirsujak	100-199	-	-	-	-	-	-	2	-	-	-
	(Wakeham Bay)											
	Kujuarapik	100-199	1	-	6	6	13	7	4	4	-	2
	(Great Whales)	200-299	-	-	2	-	1	-	3	1	1	-
		400-499	-	-	-	-	-	-	1	-	-	-
	Kuujjuaq (Fort Chimo)	100-199	1	-	-	-	-	-	-	-	-	-
	Lac Simon	100-199	-	-	-	-	3	1	-	1	-	-
		200-299	-	-	-	-	1	-	-	-	-	-
	Maniwaki (River Desert)	100-199	-	-	-	-	-	-	-	1	-	-
	Manouan	100-199	-	-	1	-	1	1	-	1	-	-
	Mistassini	100-199	2	4	5	4	28	26	29	29	7	10
		200-299	1	-	1	-	1	1	3	3	-	-
		300-399	-	-	1	-	-	-	-	-	-	-
	Obedjinan	100-199	-	-	-	-	-	3	3	2	-	2
		200-299	-	-	-	-	-	1	-	-	-	-
	Paint Hills (Wimindji)	100-199	-	-	-	1	-	-	-	2	-	-
		300-399	-	-	-	-	-	-	1	-	-	-
	Puvirnituq	100-199	-	-	-	-	1	-	-	-	-	-
	(Povungnituk)											
	Quartaq (Koartak)	100-199	-	-	-	-	2	2	1	2	1	-
	Rupert House	100-199	-	-	-	-	2	-	2	1	-	-
	(Waskagonish)											
	Salluit (Sugluk)	100-199	-	-	2	6	10	11	11	5	-	-
		200-299	-	-	-	1	-	3	1	-	-	-

TABLE 9A
TABLEAU 9A

Region Région	Community Collectivité	Level (ppb) Niveau (µg/L)	< 1 Year < 1 an		1-14 Years 1-14 ans		15-45 Years 15-45 ans		46-65 Years 46-65 ans		> 65 Years > 65 ans	
			M	F	M	F	M	F	M	F	M	F
Quebec Québec	Schefferville	100-199	-	-	-	-	2	4	5	2	3	-
		200-299	-	-	-	-	-	-	1	-	-	-
	Waswanipi	100-199	-	-	-	-	14	9	4	9	5	4
		200-299	-	-	-	-	1	-	-	2	-	-
		300-399	-	-	-	-	-	1	-	1	-	-
		500-599	-	-	-	-	-	-	-	1	1	-
		600-699	-	-	-	-	-	-	1	-	-	-
Ontario	Attawapiskat	100-199	-	-	-	-	-	-	1	-	-	-
	Deer Lake	100-199	-	-	-	-	1	-	3	-	1	1
	Eagle Lake	100-199	-	-	-	-	-	1	-	-	-	-
	Grassy Narrows	100-199	3	1	4	5	20	8	4	4	1	-
		200-299	-	-	1	2	5	1	1	-	2	-
		300-399	-	-	-	-	-	-	2	-	-	-
		400-499	-	-	2	-	2	-	1	-	-	-
		600-699	-	-	-	-	-	1	-	-	-	-
	North Spirit Lake	100-199	-	-	-	-	-	-	-	-	1	1
	Ogoki Poste	100-199	-	-	-	-	-	-	-	-	1	-
	Poplar Hill	100-199	-	-	-	1	3	1	-	1	-	-
		200-299	-	-	1	-	-	-	-	-	-	-
	Whitedog	100-199	1	-	-	2	14	5	11	2	2	1
		200-299	-	-	-	-	1	2	-	-	-	-
		300-399	-	-	-	-	1	-	-	-	-	-
		600-699	-	-	-	-	1	-	-	-	-	-
Manitoba	Cross Lake	100-199	-	-	-	-	-	-	-	-	1	-
		200-299	-	-	-	-	-	-	-	-	1	-
	Dakota Plains	200-299	-	-	-	-	-	1	-	-	-	-
	Fox Lake (Manitoba)	100-199	-	-	1	-	-	-	-	-	-	1
	Lac Brochet	100-199	-	-	-	-	-	-	-	1	-	-
	Nelson House	100-199	-	-	-	-	1	1	-	-	-	-
	Pauingassi	100-199	-	-	-	-	2	-	2	-	-	-
	South Indian Lake	100-199	-	-	-	-	-	2	1	1	1	1
	York Landing	100-199	-	-	-	-	1	-	-	-	-	-

TABLE 9A
TABLEAU 9A

Region Région	Community Collectivité	Level (ppb) Niveau (µg/L)	< 1 Year < 1 an		1-14 Years 1-14 ans		15-45 Years 15-45 ans		46-65 Years 46-65 ans		> 65 Years > 65 ans	
			M	F	M	F	M	F	M	F	M	F
Saskatchewan	Kahkewistahaw	100-199	-	-	-	-	-	1	-	-	-	-
	Kinoosao	100-199	-	-	-	-	-	-	-	-	1	-
	Lac La Ronge	100-199	-	-	-	-	-	1	-	-	-	-
	Little Black Bear	100-199	-	-	-	-	-	1	-	-	-	-
	Mistawasis	100-199	-	-	-	-	-	1	-	-	-	-
Alberta	Blood	100-199	-	-	-	-	-	-	-	1	-	-
British Columbia Colombie - Britannique	Necoslie	100-199	-	-	-	-	-	1	-	-	-	-
	Nimpkish	100-199	-	-	-	-	-	-	-	1	-	-
	Sheshaht (Tsahaheh)	100-199	-	-	-	-	1	-	-	-	-	-
Northwest Terrirories Territoires du Nord-Ouest	Arctic Bay	100-199	-	-	-	-	-	1	-	-	-	-
	Broughton Island	100-199	-	-	-	-	3	1	-	-	-	-
	Cambridge Bay	100-199	-	-	-	-	-	1	-	-	-	-
		200-299	-	-	-	-	1	-	-	-	-	-
	Clyde River	100-199	-	1	-	-	2	-	2	-	-	-
	Fort Franklin	100-199	-	-	-	-	-	-	-	1	-	-
	Fort Good Hope	100-199	-	-	-	-	-	1	-	-	-	-
	Igloolik	100-199	-	-	-	1	2	4	-	2	-	-
		200-299	-	-	-	-	1	-	-	-	-	-
		300-399	-	-	-	1	1	1	-	-	-	-
	Rae Lakes	100-199	-	-	-	-	-	-	1	-	-	-
	Resolute Bay	100-199	-	-	-	-	1	-	-	-	-	-
		200-299	-	-	-	-	1	-	-	-	-	-
	Sanikiluaq	100-199	-	1	-	-	-	-	-	1	-	-
	Tuktoyaktuk	100-199	-	-	-	-	-	2	-	-	-	-
Yukon		0	-	-	-	-	-	-	-	-	-	-

TABLE 10
TABLEAU 10

MOTHER/CHILD CORD BLOOD SAMPLING RESULTS
JANUARY 1, 1983 TO DECEMBER 31, 1996 BY REGION AND LEVEL

RÉSULTATS DES ANALYSES DE SANG MATERNEL ET DE SANG DU CORDON
DU 1er JANVIER 1983 AU 31 DÉCEMBRE 1996
PAR RÉGION ET NIVEAU

Region Région	Total Individuals Nbre total de personnes	0-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	ppb µg/L
Quebec Québec												
Mother Mère	-	-	-	-	-	-	-	-	-	-	-	
Child Enfant	15	15	-	-	-	-	-	-	-	-	-	
Ontario												
Mother Mère	96	93	3	-	-	-	-	-	-	-	-	
Child Enfant	443	432	8	2	1	-	-	-	-	-	-	
Manitoba												
Mother Mère	44	41	2	-	-	1	-	-	-	-	-	
Child Enfant	40	40	-	-	-	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest												
Mother Mère	37	32	2	1	-	1	1	-	-	-	-	
Child Enfant	6	4	2	-	-	-	-	-	-	-	-	
Total Mother Total Mère	177	166	7	1	-	2	1	-	-	-	-	
Total Child Total Enfant	504	491	10	2	1	-	-	-	-	-	-	

TABLE 10A
TABLEAU 10A

MOTHER/CHILD CORD BLOOD SAMPLING RESULTS
TO DECEMBER 31, 1996 BY REGION AND LEVEL

RÉSULTATS DES ANALYSES DE SANG MATERNEL ET DE SANG DU CORDON
AU 31 DÉCEMBRE 1996
PAR RÉGION ET NIVEAU

Region Région	Total Individuals Nbre total de personnes	0-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	ppb µg/L
Quebec **Québec**													
Mother Mère	138	129	7	2	-	-	-	-	-	-	-	-	
Child Enfant	944	593	132	78	44	39	22	12	9	5	9	1	
Ontario													
Mother Mère	951	899	29	13	5	2	1	1	1	-	-	-	
Child Enfant	1664	1444	123	38	18	15	15	5	3	-	3	-	
Manitoba													
Mother Mère	135	129	5	-	-	1	-	-	-	-	-	-	
Child Enfant	119	113	4	-	1	1	-	-	-	-	-	-	

TABLE 10A
TABLEAU 10A

Region Région	Total Individuals Nbre total de personnes	0-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-199	200-299	ppb µg/L
British Columbia Colombie Britannique													
Mother Mère	29	28	1	-	-	-	-	-	-	-	-	-	
Child Enfant	65	63	2	-	-	-	-	-	-	-	-	-	
Northwest Territories Territoires du Nord-Ouest													
Mother Mère	63	48	7	2	1	2	1	-	2	-	-	-	
Child Enfant	40	11	12	8	2	3	-	1	-	1	2	-	
Total Mother Total Mère	1316	1233	49	17	6	5	2	1	3	-	-	-	
Total Child Total Enfant	2832	2224	273	124	65	58	37	18	12	6	14	1	